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THE SURGERY OF TRAUMA.[1]

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AMBROISE PARÉ records that "while the King of France was besieging Turin the besiegers and the besieged mutually believed that their enemies had poisoned their cannon balls, so cruel and intractable were their wounds; but after the taking of the city the soldiers of both parties met and then they saw that their own clean and unpoisoned cannon balls had made the same cruel wounds". And apropos of cauterizing these wounds with boiling oils, he makes the following statement:

> I, also, followed the practice of drenching wounds with boiling oil until all my oils were expended. On the night of which this happened, I dressed my wounded soldiers with Oil of Roses and Turpentine with whites of eggs. I went to bed much oppressed, with the apprehension that all those poor fellows would be found in the morning poisoned and dead. I arose therefore betimes, and learnt to my infinite surprise and pleasure that they had slept well and were without pain and swelling or redness about their wounds, while those that had been cauterised by hot oil had great fever and swelling and excruciating pain.

This age-old practice of cauterizing wounds thus came to an end in the middle of the sixteenth century. Until the close of the eighteenth century it was still generally believed that natural healing of wounds occurred through the agency of laudable pus. Although this had been denied as early as the thirteenth century, the denial had never been generally accepted, and as late as 1764 Samuel Mihles wrote these words:

> The progress of a wound is distinguished into four stages. So long as the hæmorrhage or ichorous discharge continues, the wound is said to be crude. When uniform laudable matter appears and takes off all the dead extremities of the fibres and muscles which have been corrupted by the air, it is said to be in a state of digestion, and when the surface of the wound appears of a clean red, without inflammation, and its cavity begins to diminish by the sprouting ends of the fibres, the wound is said to be in a state of incarnation, and finally, after the wound is filled by the skin and cuticle extending themselves over the margin, by degrees spreads over the whole wound, then the wound is said to be in a state of cicatrization which completes the cure.

Soon after this, John Bell in 1795 stressed the fact that healing could, and did, occur by primary intention. This was his answer to the adherents of the laudable pus theory:

> When a modern surgeon allows himself to talk about the mundifying, incarning and cicatrizing of wounds, or directs how to fill the wound up with good and sound flesh and keep it to a fair and even level with the adjacent skin, he but proclaims his own ignorance of the properties of the living body. When we can prevail upon ourselves to renounce this parade of idle words and to resign our supreme office of assisting nature in mundifying, incarning and cicatrizing, we shall cast behind us a jargon of words and a farrago of useless drugs which belong to the times of ignorance, and we shall find our duties happily reduced within the narrowest bounds, i.e. of saving the patient from immediate bleeding and of laying the wounded parts so cleanly, so neatly and so evenly in contact with each other that they may adhere. The rest we leave to nature.

Primary closure of wounds and the saving of skin edges now became an established practice and the flap amputa-

[1] Read at a meeting of the Queensland Branch of the British Medical Association on April 1, 1949.

tion began to replace the guillotine method. So far we have seen the belief of the poisoning of wounds dispelled, the time-honoured treatment by cauterization abolished, and primary closure of wounds and flap amputation introduced. Primary closure was all very well in the "clean" case; but the problem of infection remained and in point of fact is still one of the main worries of the traumatic surgeon.

In the early part of the nineteenth century the importance of the part played by foreign material in a wound was realized. Not only was an extraneous foreign body deemed to be harmful, but so also was devitalized tissue. Accordingly, Joseph Desault and John Larrey, as a result of this experience in the Napoleonic War, introduced surgical excision of damaged tissue and *débridement*. *Débridement* is used here in its original sense of incision for the purpose of drainage. These men also taught the necessity of treating wounds as soon as possible after injury. Incidentally, Larrey claimed that traumatic or gas gangrene was due to an upset of the sympathetic nervous system, and although this conception is far from the truth, the importance of muscle ischæmia, due to arterial damage or spasm in gas gangrene, has been appreciated only within recent years.

The next step forward came when complete immobilization of the injured limb was advocated by Pirogoff and Ollier, and at about the same time antiseptic surgery was introduced. Experience of the past was straightway forgotten. The practice of excision and drainage was discarded and reliance was placed wholly on cleansing the wound with antiseptics and promoting healing by immobilization. It was not until the war of 1914-1918 that wound excision was revived; but now, with encouragement from the great advance that general surgery had made with the more recent aseptic era, primary closure again became popular and, of course, with disastrous results.

It seemed now as if in desperation the pendulum swung to the opposite extreme, and Carrel introduced the technique of continuous irrigation of the wound after wide excision. Improved results soon justified this method. Towards the end of the first World War the closed plaster technique of Winnett Orr was being used for chronic osteomyelitis with considerable success, and in the Spanish Civil War of 1936-1939 this technique, somewhat modified, was used by Trueta as a method of primary treatment of war wounds. And so the last great military conflict opened with the standard treatment of wide excision, *débridement*, gauze packing and "closed plaster". The results of this method of treatment were highly satisfactory, and it remains largely the method of choice today, with the exception that since the introduction of penicillin and allied antibiotics, primary closure of wounds may be carried out more often. Here let me issue a note of warning in case we should commit the same folly as our predecessors and forget those principles of wound treatment which have been so painfully and laboriously learned.

In penicillin we have a new, powerful weapon, a weapon which in many ways is revolutionary, but a weapon which has its limitations. We know that such chemotherapeutic agents, at the moment at any rate, are not effective against all organisms, and we know, too, that to be effective they must reach every portion of the wound and be present there in an adequate bacteriostatic concentration. What would be the end result of a wound in which inadequate excision had left islands of dead tissue as organismal sanctuaries in a lake of penicillin, a wound in which inadequate incision allowed the continuation of sufficient tension to prevent the influx of penicillin, or a wound in which arterial damage or spasm had rendered a muscle group ischæmic and therefore a prey to the organisms of gas gangrene?

It is only when the fundamental principles of wound treatment have been fulfilled that primary repair may be contemplated. There can be no room for doubt that primary repair of tissues gives an enormously better anatomical and functional result than does secondary repair, and it should therefore be the surgeon's aim to carry out as a primary procedure tendon repair, nerve suture, internal fixation of fractures, skin grafting and so on. Modern chemotherapy offers an almost limitless scope of primary reparative surgery, provided—and again I emphasize the point—that all the fundamental principles of wound treatment are religiously observed.

Briefly stated, these principles are as follows: adequate resuscitation before, during and after operation; thorough mechanical cleansing of the wound and surrounding area (a cake of soap and some water are all that is required); removal of all inert foreign material; complete excision of all devitalized tissue; adequate *débridement*; the elimination of all dead spaces by accurate reduction and fixation of fractures; absolute hæmostasis; complete closure of all raw areas by suture if it can be carried out without tension, otherwise by skin grafting with local rotation flaps, distant pedicles or free split skin grafts; complete and uninterrupted immobilization; and finally the administration of penicillin, which should be commenced on the patient's admission to hospital.

With reference to these principles, they apply no less to full thickness burns than to open wounds. When it is known that the full thickness of skin is involved, treatment by total excision and immediate skin grafting is the method of choice. Most wounds are bacteriologically contaminated, but when they are treated early, frank infection should not occur. The occurrence of infection in such wounds is due to the presence of ischæmic tissue. Perhaps I have referred too briefly to the all-important principle of complete excision of devitalized tissue. It may be a very difficult matter when it comes to the point. Apart from the usual methods of determining dead tissue at operation, we can be guided to some extent by the nature of the trauma. I feel there is too great a tendency to disregard this. A compound fracture of the tibia may be caused by a linear force, another by a fall of heavy masonry, yet both may appear somewhat similar on inspection. In the first of these we know there will be little avascular tissue and little likelihood that dangerous tension or vascular spasm will develop. In the second, the crush injury, more widespread damage with subsequent swelling and death of muscle and skin is much more likely to occur. Similarly, the resulting devitalization and loss of function in a hand are considerably greater after a crush between rollers, even though no fracture has occurred, than in the case of multiple compound fracture caused by a sharp instrument, and greater still when the hand is caught between hot rollers. Doubt as to the extent of surgical excision must always accompany the injury caused by a crushing or explosive violence, and fewer liberties may be taken with regard to primary repair. So much for the treatment of open wounds.

As far as closed injuries are concerned there has not, of course, been a progressive march of events, and I will mention only a few of the more conspicuous advances.

Pseudarthrosis following fractures was once very common, but is now a comparative rarity. This is due in the main to the advent of bone grafting and internal fixation by the use of non-toxic metals, such as stainless steel, vitallium and tantalum.

Ollier, Barth, Axhausen and McEwan are among those whom we have to thank for placing bone grafting on a firm footing. Since their day many improvements in technique and in types of graft have been introduced; but problems and difficulties still exist. The problem of the separate operation to obtain the bone for grafting and the limited amount of cortical and cancellous bone which is available without serious mutilation may deter the timid from the attempt and dishearten the brave by failure of the graft. There is, however, a relatively simple answer to these particular problems and that is the bone bank. Bone banks have been referred to as luxuries; if that is so, they are essential luxuries. Every accident and orthopædic centre should have access at any time to a bank of stored bone. Many bone-grafting operations fail as a direct result of an inadequate bulk of graft, and many others, though excellent in conception, are impracticable for the same reason.

The sympathetic nervous system has only recently been receiving the attention which is its due. It is now becoming evident that arterial spasm is the underlying disorder in

many hitherto obscure lesions. We are all only too familiar with the forearm and hand which have been twisted and distorted by degenerated and fibrosed muscles and horribly scarred by the surgeon's incisions of desperation, all of which go to make up the picture of a fully fledged Volkmann's contracture. More localized forms of arterial spasm are seen in those cases of flexion deformity of the fingers due to ischæmic contracture of the lumbrical muscle alone. Spasm of the collateral circulation may determine the loss of a limb when its main vessel has been damaged. If to these we add the various types of post-traumatic osteodystrophy we find a group of lesions in which treatment by direct or indirect attack on the sympathetic nervous system has produced some dramatic cures—and, I add, some equally dramatic failures.

It is important to realize that plastic surgery can do much to restore lost function to extremities, as well as to improve one's facial appearance. If this is kept in mind, a primary reparative procedure may be modified in order to set the stage for a later reconstruction. There is little to choose between a hand which has a thumb but no opposing fingers and one which consists of four fingers and no thumb. In both the important function of prehension is virtually lost. To restore to these hands at least the basic opposition digits—that is, a finger and a thumb—many ingenious reconstructive operations have been devised, and the success of these procedures may be dependent on the skill and foresight of the surgeon who performed the primary operation. To restore a lost digit, one of the remaining digits may be transposed to the site of one that is missing, a finger of the other hand or a toe may be used as a free graft, or an entirely new finger may be constructed from living tissues, as in the skin tube and cancellous bone graft method. Although some of these procedures can be regarded as extravagances of surgical virtuosity, they nevertheless represent a definite advance in the art of traumatic surgery, and they are certainly in keeping with what should be the motto of the traumatic surgeon: "Functional results before anatomical results."

If I was asked what I considered to be the greatest advances in the surgery of trauma within recent years, I should say the development of methods of resuscitation, the tendency toward the organization of special clinics and departments, and the emphasis now placed on rehabilitation.

Resuscitation is now so well known and universally adopted that no further discussion is necessary. I should like to give you one example of its application.

The story concerns a military patient received in a base hospital in Scotland. On the Normandy beaches he received a blood transfusion, his ruptured spleen was removed and his torn gut was sutured. After a second blood transfusion he was flown back to England, and then travelled by train to Scotland. In transit he did not miss one of his four-hourly injections of penicillin. He arrived in Edinburgh in first-class condition twenty-four hours after being wounded.

This is surely a tribute, not only to resuscitation and the surgeon, but to military organization.

As far as specialization is concerned, I am aware that the grouping of special types of cases, or an increase in the number of already existing specialties, is a controversial matter; but in some cases I am sure there is no room for doubt. Can anyone deny that there has been greater *propter hoc* improvement in the treatment of fractures since the introduction of special fracture clinics?

Hand surgery is another specialty which has justified itself. Until a few years ago it was the common belief that when a flexor tendon of a finger was divided within its digital sheath, the function of flexion could not be restored and that the best treatment was amputation of a useless finger which did no more than get in the way.

Today good results are being obtained from primary or delayed suture and free tendon grafts, and every reported series of cases shows an ever-increasing percentage of good functional results. This state of affairs, which gives so much hope to the artisan, has been brought about by the energies of a mere handful of men, notable among them being Sterling Bunnell, Michael Mason and Sumner Koch.

It was they who in America took hand surgery away from the house surgeon and put it in its rightful place under the care of skilled and experienced surgeons working in specially created departments or even special hospitals, set up to deal solely with hand injuries. There are other special units to which I will refer later.

I now come to the third of the important advances mentioned—rehabilitation.

Medical literature of the past tells us little of after-treatment and final functional results. In fact, we have been left with the impression that most injuries particularly fractures, were necessarily followed by some degree of permanent disability. This is certainly not the case today, and yet all the advances in treatment which have been mentioned so far are hardly sufficient to be entirely responsible for this present-day improvement. What, then, is the remaining missing factor? It is the fact that our energies are being directed toward treatment, not of the isolated wound, but of the individual who bears that wound. This is a vast subject, and I am afraid that I shall not have time to give anything more than a brief sketch of it.

It was not until Sir Robert Jones began his activities in this sphere in the first World War that any real contribution to rehabilitation had been made. He foresaw the great number of broken-limbed and broken-spirited men that would arise as an aftermath of war. As a result of his foresight and energies arose Shepherd's Bush, the first concept of large-scale rehabilitation centres which are now being established throughout the world. In spite of the tremendous amount of excellent work that was done, and in spite of the obvious significance of rehabilitation, quite apart from the injuries of war, Shepherd's Bush and the lessons it taught were doomed to be largely forgotten during the next twenty years.

However, in the 1930's it was becoming evident that rehabilitation was to be reborn, and the second World War, in providing the urgent necessity for restoring the injured to maximal functional capacity in the minimal space of time, precipitated this rebirth. Rehabilitation in large centres as conceived by Sir Robert Jones was this time championed by Sir Reginald Watson-Jones.

Let me read a short story of his, from "Rehabilitation in the Royal Air Force"; behind it are the full story and significance of rehabilitation.

> In November, 1940, a patient was admitted to hospital for the displacement of a semi-lunar cartilage. In August, 1941, he was still in hospital. Treatment had been continued for ten months, but the incapacity was still total. Why was recovery so long delayed? What complication had arisen? The diagnosis was correct and a torn cartilage had been removed. The operation had been skilfully performed and the wound was soundly healed. The ligaments were undamaged—there was no synovial effusion and no arthritis. It is true that the muscles were weak, and despite daily massage and two manipulations, the joint was stiff. The gait was slow and hesitant—he could not run—he had never tried to run.
>
> The house surgeon blamed him for lack of co-operation and said that he was depressed, disinterested, resentful and "a poor type." It seemed odd that he should be a "poor type" because he was an Air-Gunner, a volunteer for Air-crew duties. He was certainly depressed, but not without good reason, for after 10 months the disability was more complete than on the day of admission. He was disinterested because "nobody takes any notice and it looks as if it is hopeless." He was resentful because he could not believe that the fault was his. Had he not been told that "the nerve to his knee was cut!"
>
> He was transferred to an orthopædic rehabilitation centre—he saw the sky, the sea, the open-spaces. For months he had seen stone walls—the stone walls of hospital buildings, the stone walls of massage departments, the stone walls of many corridors. Now the wind blew in his face and he felt the spring of the turf. He no longer slept in a ward but in a bedroom—there were no nurses. He could smell no drugs or antiseptics. The rooms were brightly decorated and there were flowers. The menu was varied. There was an atmosphere of well-being and contentment.
>
> The Medical Officer who examined him spoke of adhesions and muscle control, explained the symptoms

that worried him and enquired about his home and family. It seemed unusual, but he appreciated it. He was taught exercises and put with a group of men who were boisterous and happy. He sensed the optimism, and was re-assured. He was taught to walk without a limp and was congratulated. He sunbaked—when he was idle he was told firmly that he must work. He learned to mark time on tip-toe—to do it more quickly—to move one inch with each step, and before realising what had happened he found he was running. There was a sparkle in his eye. He worked in the gymnasium, played on the fields, swam in the pool, cycled on the track, attended lectures, went to concerts. Time raced past, for he was busy. He became bronzed and fit. He laughed and was full of the joy of living. In seven weeks he returned to his unit and to full duty. "The nerve to his knee" was forgotten. Ten months, total disability—seven weeks, full recovery.

That is the story of rehabilitation in one air-gunner. Let us attempt to analyse this story.

The direct cause of the patient's apparent permanent disability was a psychological reaction to the fear of disablement. Such a reaction is often due to other fears: the fear of being down-graded in industrial skill, the fear of domestic difficulties as a result of loss of wages, the fear of returning to the same machine which caused his accident and so on.

The statement that he was taught exercises bears some comment. Think for a moment of a case of, say, a storeman and packer who sustains a partial tear of his supraspinatus tendon. He is sent to a physiotherapy department of a general hospital—that is all you can do with him. Here for one hour a day he has his shoulder baked beneath an impressive array of electric light bulbs and he is encouraged to creep up the wall with his finger tips, and to turn and re-turn a dummy helm. Undoubtedly, this hour of treatment is beneficial, but what then? With his hands in his pockets he proceeds to while away the remaining hours of the day in idleness of muscle and idleness of mind, perhaps to listen to wild tales of his neighbour's paralysed arm or to more malicious stories of how to get more compensation. The next day his shoulder is as stiff and as painful as ever, perhaps more so. He may become another rear gunner. Do not think that I am condemning physiotherapy—far from it; used in the right case and at the right time, it is an essential part of rehabilitation, but it is not a cure-all, nor is it the whole picture. It is one link in a chain of treatment, and without the other links it cannot be as effective.

The story tells us that the gunner played a game of hand-ball and became an enthusiast. Hand-ball comes under the heading of gymnastics or physical training or recreational therapy. The gymnasium with indoor and outdoor sports has a greater interest-holding capacity than physiotherapy, and another advantage that at least half the day can be spent in its activities. Localized purposeful movements and general physical exercises are combined with numerous mental factors so important in the general treatment. There is no need to explain the value of such things as example set by fellow sufferers, or the determination that can be developed by jealousy of the other fellow's progress, or provoked by shame of one's own laziness. There is no need to talk about the general atmosphere which can be created by a good physical training instructor and how far that goes in treatment. It is remarkable what rapid strides are made and how rapidly a chronically painful knee can be forgotten after a strenuous game of hand-ball, particularly when a little ingenuity and simple psychotherapy has been shown in choosing the sides and in giving them appropriate names.

The story then goes on to say that "when he was idle he was told firmly that he must work". Here is occupational therapy. This type of treatment has the advantage that the whole of the day can be spent in the interest of the patient. If the work is well chosen, it adds to prescribed purposeful movements an absorbing interest and possibly remuneration. It is generally agreed that a job of work to do combined with periods of recreation and diversion are necessary to an ordinary healthy and happy existence. How much more necessary, then, are these things to those who are suffering from bodily and mental injury? One of the more elaborate features of occupational therapy is the sheltered workshop. Here injured skilled workers are employed in productive work on ordinary industrial machines which have been slightly adapted to suit their particular disability. These adaptations are gradually removed as the disability diminishes. There could be no better conception of rehabilitation than the picture of an injured man returning to work the moment he leaves hospital, or even before, despite splints and despite disability, curing himself by his own efforts, but guided throughout by a team of experts.

One other aspect of rehabilitation remains to be mentioned. You will notice that the gunner was asked about his home and family. It would be unreasonable to expect a man to indulge in any of these activities if he was worried about his own future and the future of a family who were dependent upon him. It is only when the medico-social workers have ironed out his particular difficulties by obtaining domestic assistance of all kinds, by arranging holidays, by interviewing his employer and employment bureaux regarding his future possibilities *et cetera*, that he can become an enthusiast about his full-time job of getting better.

The word "rehabilitation" is perhaps an unfortunate one, for the story of the gunner may well imply that we must first allow the injured man to become disinterested and dispirited, his muscles to atrophy, and his domestic affairs to collapse before we begin to rehabilitate him. A greater misconception could not exist. It is in the prevention of these things that rehabilitation plays its greatest part. The methods adopted for prevention are, of course, precisely the same as those which have been described for cure.

In summary, rehabilitation begins with first aid at the site of injury, it is continued in hospitals and specially equipped centres by a team of highly trained personnel in the fields of medicine, medico-social workers, with physiotherapy, physical training and occupational therapy including recreation and diversion. It does not cease until the injured man has been restored to his original status, or to his maximal level of functional ability, mental equilibrium and social standing.

Although I have been referring to rehabilitation in relation to trauma, this is not its only field of usefulness. I doubt if there is any branch of medicine which could not use a rehabilitation centre with considerable advantage. I have already indicated that rehabilitation is designed, not so much for the treatment of any specific disease, as to maintain or to restore to an individual a generally healthy state of mind and body. I am certain that the number of chronic drug takers and other human derelicts could be substantially reduced by the universal use of rehabilitation centres.

The number of tuberculous and psychiatric patients requiring rehabilitation is probably equal to that of accident patients; yet this latter group alone is large enough to justify the setting up of rehabilitation units.

I wonder if many of us realize the full significance of accidents?

The foundation of civilization is based on labour. The injured labourer therefore becomes a liability to his dependants, to his employer and to society as a whole. The percentage incidence of industrial accidents is steadily increasing, and apart from the personal factors concerned, the resulting domestic hardships and the overall loss to society are enormous.

B. K. Rank gives a graphic account of industrial loss in the following statement:

> . . . the amounts paid in compensation are generally estimated at only one-fifth to one-quarter of the total cost of industrial injuries to the community. Compensation costs in this State [Victoria] were approximately one million pounds last year. The problem is of far greater magnitude in America than in other countries. In the year 1941, for example, 1,500,000 were injured in industry, 70,000 permanently disabled, and there were 18,000 fatal employee accidents. The total loss in man hours and cost would have built and paid for a fleet of 26 first-line battleships and 9,000 Flying Fortresses. It represented a bottle-neck far more serious than the slowing-down by strikes and shortages.

Now those figures, enormous as they are, are only part of the story. For every two industrial accidents there are three domestic and four road accidents, bringing the total to astronomical levels.

Numerous factors are involved in the cause of industrial accidents. Many of them are known and much creditable work has been done to counteract them. Others are less well known and are difficult to combat. Some of the factors concerned are lighting, ventilation, hours of work and recreation, age and capabilities in relation to the job, interest in the work, general health, adequate supervision and guarding of machinery. Various bodies which have been engaged in the prevention of factory accidents have done well in confining machinery accidents to 10% to 20% of the whole. But surely something is still wrong when boys of fourteen years of age, within the first weeks of their employment, have their hands mutilated by circular saws, scarifiers and mincing machines! Such accidents are only too common. Of all such accidents 80% to 90% are caused by personal factors, and a great deal is still to be done in this respect if the present enormous accident rate of one in four of the industrial population is to be reduced.

Cooperation between the medical profession and other bodies in a scientific approach to the matter is a great necessity. How can this be done?

Conclusion.

I have indicated that practically all the advances in traumatic surgery have come as a result of treatment in organized centres. The surgery of wounds grew in military hospitals, and the treatment of fractures began to improve rapidly once fracture clinics were set up. More recently peripheral nerve units and burns units have been established. Hand surgery came into being only with the creation of special departments, and now there is the rehabilitation centre. It seems that we cannot do without this type of specialization.

Surely, then, the logical conclusion is to combine all these units into one integrated whole. This implies the setting up of hospitals to deal solely with the problem of trauma—in other words, accident hospitals. Such hospitals would be concerned with active treatment, rehabilitation and resettlement. They would create much greater incentive and opportunity to develop the academic side through clinical and scientific research. Undergraduate and post-graduate teaching would present little or no problem. Working in conjunction with other bodies concerned, such hospitals would be able to contribute something material towards the problem of the cause and prevention of industrial and transport accidents. Through rehabilitation and retraining centres, they would materially assist in the resettlement of the permanently, partially or totally disabled. In these ways and others they would become an integral part of the industrial social structure, and in so doing would render a true service to the people—a service which would far transcend in value to the individual his weekly compensation, his lump-sum settlement, or his free medicine. Such a service would play a definite part in the future prosperity of a modern community.

PORTAL HYPERTENSION AND ITS TREATMENT.

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THE present interest in portal hypertension has its origin in the original demonstration by Eck in 1877 that it is possible to anastomose the portal vein to the *vena cava* without apparent harm to the individual.

The recent work of Blakemore and Lord has given a further stimulus to this subject. They showed that it was a relatively simple procedure to anastomose the portal vein to the inferior *vena cava* by the use of vitallium tubes.

The procedure to be described in this article is that of spleno-renal anastomosis, with a thoraco-abdominal approach and a continuous silk-suture technique.

Types of Hypertension.

Portal hypertension occurs in three types: (i) the intrahepatic type, (ii) the extrahepatic type and (iii) the combined type.

The intrahepatic type is most commonly caused by cirrhosis of the liver. A second cause is thrombosis of the hepatic veins.

The extrahepatic type, commonly known as Banti's syndrome, may be congenital or acquired. The congenital variety is characterized by obliteration of the portal vein and cavernomatous transformation of the vessels in the lesser omentum leading to the liver. The acquired variety is produced by thrombosis of the portal vein, which may be caused by infection or by trauma, or may be spontaneous.

The combined variety is uncommon.

The result of portal-bed block of all of these types is engorgement of the portal system. The maximal incidence of this engorgement is dependent upon the exact site of the block. In the intrahepatic type of block, the engorgement will be throughout the whole of the portal system (Figure I). In the extrahepatic type, the engorgement

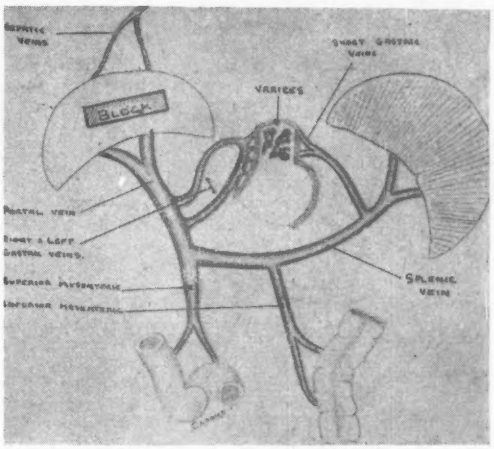


FIGURE I.
Intrahepatic type of portal obstruction resulting in generalized portal bed engorgement.

may be in the whole of the portal system (Figure II), but it may involve only a portion, such as the splenic vein, and that would therefore result in the engorgement of the spleen, *vasa brevia* and œsophageal veins (Figure III). One of the commonest manifestations of such portal engorgement is hæmatemesis from dilated œsophageal varices. It must be remembered, however, that the engorgement is throughout the portal system, and bleeding may occur from engorged vessels in the stomach or other portions of the alimentary canal.

Diagnosis.

The diagnosis of portal hypertension is usually made subsequently to the occurrence of hæmatemesis or melena. The physical examination may be significant. There may be general distension of the abdomen, or there may be fullness in the right hypochondrium, indicating enlargement of the liver, or fullness in the left hypochondrium, indicating enlargement of the spleen. There may be dilated veins coursing over the thorax and the abdomen, and there may be demonstrable free fluid in the abdomen. In the extrahepatic type, the liver will not be involved and will therefore be of normal size.

In the intrahepatic type, however, the liver may be enlarged or atrophic, depending upon the type of cirrhosis.

A further point is that with blockage of the splenic vein, the spleen will be enlarged, the liver will be normal, and there will be few, if any, engorged veins coursing over the thorax and the abdomen.

Liver function tests will be of value in differentiating the intrahepatic from the extrahepatic types. Of these, the prothrombin time estimation, the hippuric acid excretion, the bromsulphalein excretion and the flocculation tests are the most valuable. Normal results from liver function tests are suggestive of an extrahepatic block.

The serum proteins are of considerable importance, as with a normal liver the serum proteins will be normal in amount and with a damaged liver the serum proteins will be decreased in amount.

It is of particular importance to note the albumin-globulin ratio. With severe liver damage the albumin level is considerably reduced; this results in failure to retain fluid within the blood-stream, and, as a consequence of this, ascites develops. It is most important to realize this fact, as ascites as a consequence of liver failure will not be relieved by any type of anastomosis between the portal and systemic systems.

A barium bolus examination will frequently demonstrate œsophageal varices at the lower end of the œsophagus.

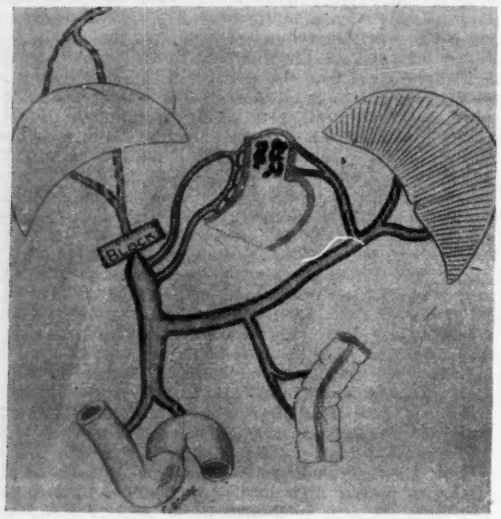


FIGURE II.

Obstruction at the *porta hepatis*. Extrahepatic type of portal obstruction resulting in generalized portal bed engorgement.

Principles of Operation.

Consideration of Figure I will show that the whole of the portal stream may be diverted by anastomosing the portal vein to the *vena cava*. Obviously this will relieve the portal hypertension. Liver function is carried on by blood reaching the liver cells in an abnormal fashion from the hepatic artery and from smaller anastomotic vessels.

It remains to be shown by observation over a number of years that this complete diversion of the portal flow is not a bad thing.

By anastomosis of the splenic vein to the left renal vein, it will be seen that a considerable proportion of the portal stream is diverted to the systemic stream. The proportion diverted is approximately 40%, but obviously this must be a very variable amount.

Splenectomy alone will cure portal hypertension resultant upon splenic vein block. Splenectomy is also an essential part of the reduction of portal hypertension by spleno-renal anastomosis, as the splenectomy itself results in a diminution of portal flow by approximately 40%.

With a porto-caval anastomosis, undoubtedly the whole of the portal flow is diverted, but this procedure is a particularly hazardous one because of the very great number of vessels in the region of the *porta hepatis*. A false move will always result in profuse hæmorrhage, which may be almost impossible to control. Attempts to control this hæmorrhage may very easily result in irreparable damage to the vital structures in the region.

The operation of spleno-renal anastomosis is technically difficult, and the same difficulties with anastomotic vessels will be encountered, but it is not so difficult as a porto-caval anastomosis, and not nearly so hazardous. If extreme difficulties are encountered, the patient can be saved by splenectomy and nephrectomy. If comparable difficulties are encountered in a porto-caval anastomosis, irreparable damage has usually been produced by this time, and the patient will die.

The relative safety of spleno-renal anastomosis compared with that of porto-caval anastomosis, taken in conjunction with the 40% diversion of flow by the anastomosis and the 40% diminution in flow by the splenectomy, renders this the most favourable operation. The only time

FIGURE III.

Thrombosis of the splenic vein. Extrahepatic type of portal obstruction resulting in "splenic pattern" type of portal bed engorgement.

a porto-caval anastomosis is indicated is when there is some anatomical contraindication to a spleno-renal anastomosis.

Technique of Operation.

The patient is placed in the right oblique position (Figure IV); in this position the renal vessels are running horizontally. An incision is made over the line of the tenth rib, extending from the mid-point of the *rectus abdominis*, back to the lateral margin of the sacrospinalis group. The tenth rib is excised, and the pleural cavity is opened. The diaphragm will be seen, and the lung will just come down into the wound. The diaphragm is incised with caution, as it is sometimes involved in a cavernomatous enlargement of the vessels so great as to resemble a neoplastic process.

When the abdomen is entered in the intrahepatic type (Figure I), the whole of the portal bed will be seen to be engorged, the portal veins will be seen to be tense, and they will have the texture of arteries rather than of veins.

If the engorgement is of the extrahepatic type, the site of the block will result in a particular pattern of

engorgement. For example, in the splenic bed type (Figure III)) the engorgement of the portal bed will be of the splenic vein, the spleen and the close tributaries of the splenic vein, such as the short gastric and œsophageal veins. The pattern of this portal hypertension having been observed, it is then necessary to take pressure readings from various points.

In the carrying out of this procedure, it is important to use a manometer of 500 millimetres' calibration, attached to which is a fine needle and a piece of rubber tubing together with a tap. This system must be filled with normal saline. A vessel is selected, the needle is inserted into the vessel, and the tap is opened immediately. It is important to realize that it is a column of saline that is being supported, rather than a back-flow of blood up the needle, which, of course, will not occur. By taking pressure readings, it may be demonstrated that there is hypertension in the whole of the portal system or in a portion of it. For example, if there is hypertension of the splenic type, a tributary in the region of the hilum of the spleen will be under increased pressure, whereas a reading taken from the right or left gastric vein will show a normal pressure, thus demonstrating that the block is somewhere in the splenic vein.

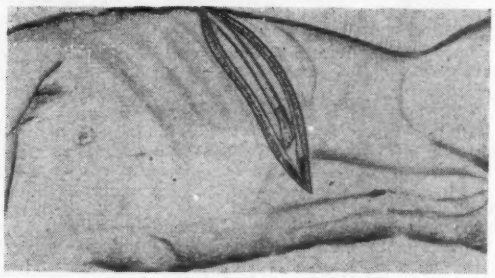


FIGURE IV.
The site of incision. Showing right oblique position and incision in line of tenth rib.

By consideration of Figure I the various points will be seen at which pressures should be taken. In the interpretation of the pressure reading, the very variable manner of termination of the gastric and of the superior and inferior mesenteric veins must be constantly in mind.

If it is demonstrated that there is merely a blockage of the splenic veins, splenectomy is all that is indicated. If, however, there is a block of the greater portion of the portal system, splenectomy will be inadequate and an anastomosis will be indicated. The spleen should be removed by ligation of the terminal branches of the vessels within the hilum of the spleen. This having been done, the peritoneum on the posterior surface of the abdomen in front of the kidney is incised (Figure V), the false capsule of the kidney is exposed and the renal vessels are cleared (Figure VI). This is an avascular plane and much easier to dissect than the plane between the pancreas and the false capsule of the kidney.

By careful dissection the renal vessels are exposed, almost to the mid-line. This having been done, the tail of the pancreas is picked up, and the connective tissue on its posterior surface is dissected off. This will reveal the splenic vein, coursing along the posterior aspect of the pancreas, and its tributaries, which are very numerous. These must be divided with great care and ligated and an adequate length of the splenic vein obtained (Figure VII). This is a difficult part of the operation. An adequate length of the splenic vein having been obtained, the false capsule of the kidney is cut so that the splenic vein may be approximated to the renal vein. The renal artery is then clamped with a clamp as in Figure VII. Next, the kidney having been emptied of blood, the renal vein is clamped; the ovarian or testicular vein should also be clamped. Then the renal vein is opened transversely, and the splenic vein is anastomosed to it by a continuous suture of 0000 "Mersilk", an everting stitch being used.

It is not necessary to deliver the kidney to perform this anastomosis, nor is it necessary to maintain a circulation throughout the procedure, as was done in the case described, as the kidney can be clamped off for an adequate length of time without damage. It is preferable to leave the kidney *in situ*, as this is a safeguard against a thrombosis occurring at the site of the anastomosis. This has been shown by Blalock in a series of experiments on dogs.

It is preferable to avoid damage to the testicular or ovarian vein, but should this be in the way, it may be divided safely. Should damage occur to the splenic vein, so that it cannot be used, then another tributary of the portal system should be selected. Quite large vessels may be found, so that one of these can be utilized to make an

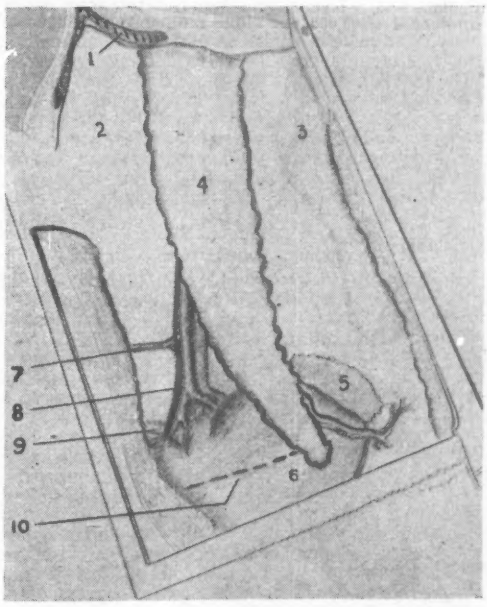


FIGURE V.
Structures exposed after removal of spleen. 1. Left rectus. 2. Transverse mesocolon. 3. Stomach. 4. Pancreas. 5. Adrenal. 6. Kidney. 7. Testicular (ovarian) vein. 8. Renal vessels. 9. Ureter. 10. Incision in false capsule.

anastomosis to the renal vein or to the ovarian or testicular vein. If this is impossible, then the operation may be terminated at this stage. The patient will be considerably benefited by the splenectomy, and at a later stage a porto-caval anastomosis may be considered. Should a porto-caval anastomosis be deemed advisable at a later date, the patient will be in an infinitely better condition for such a procedure and the result will be correspondingly improved by the splenectomy.

The diaphragm is reconstructed with interrupted thread sutures. The ribs are approximated by a continuous chromicized suture. The muscles are approximated by chromicized sutures, and the skin is closed with thread.

During this procedure, it is essential to have a blood transfusion running. The patient should require no more than four pints of blood, although at times this will be exceeded. It is not advisable to use heparin, as with the considerable flow obtained through the portal anastomosis and the flow through the renal vein from the kidney, the chances of thrombosis are not great, whereas the chances of complications following the use of heparin are considerable.

Report of a Case.

A male, aged twenty-three years, who gave a history of dyspepsia for a number of years, was admitted to Prince Henry's Hospital on November 19, 1947, suffering from hæmatemesis. His hæmoglobin value was 40%. A provisional diagnosis of peptic ulcer was made and a blood transfusion commenced. To improve his condition seven pints of blood were required; even so his hæmoglobin value was still only 64% on November 25, 1947. The patient was kept to a strict ulcer regimen, but continued to have repeated hæmatemeses requiring many transfusions (11 pints).

When he was examined in consultation on January 23, 1948, it was observed that the liver and spleen were palpable and there was evidence of some free fluid in the abdomen. There were no enlarged lymph glands. The heart was normal, the blood pressure was 130 millimetres of mercury (systolic) and 55 millimetres (diastolic), and the nervous system was normal.

Investigations revealed that the blood after repeated transfusions had a hæmoglobin value of 80%, the red blood cells numbered 4,300,000 per cubic millimetre, the leucocytes

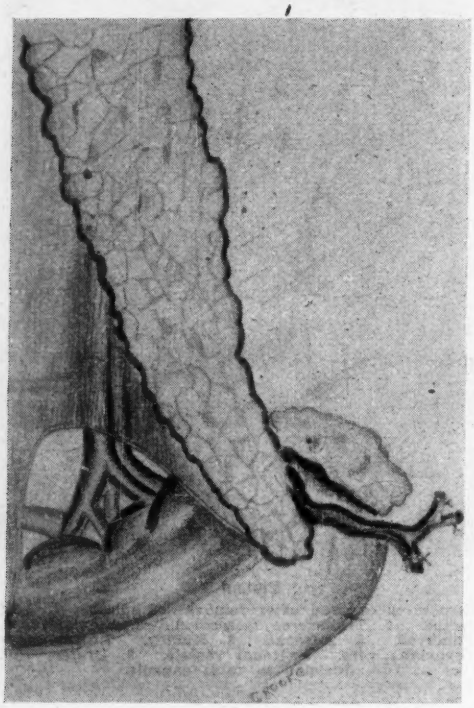


FIGURE VI.

Dissection of false capsule to expose renal vessels.

5200 per cubic millimetre and the platelets 136,000 per cubic millimetre. The total amount of serum proteins was 6·3 grammes *per centum*, made up of albumin 3·4 grammes and globulin 2·9 grammes *per centum*. The bilirubin content was 0·6 unit (normal 0·2 to 1·0 unit). The bleeding time was 10·5 minutes. The prothrombin time was 85% of normal. The hippuric acid excretion was 2·8 grammes (normal 3·0 or more grammes). The alkaline phosphatase level was 24·7 units (normal three to 13 units). The blood urea content was 23 milligrammes *per centum*. The reaction in the cephalin flocculation test was "four *plus*". The result of the Kline test was negative. The fæces contained occult blood only. Barium meal examinations carried out previously had shown no evidence of ulceration.

In view of the enlargement of the spleen and liver associated with ascites, and the absence of evidence indicating disease of the hæmatopoietic, digestive, cardiac or renal systems, it was considered that the patient was suffering from cirrhosis of the liver with resultant portal hypertension.

It was decided for reasons already indicated that a splenorenal anastomosis would be performed.

This was done on March 23, 1948. The patient had an uneventful convalescence, being out of bed on the fifth post-operative day and discharged from hospital, well, to a convalescent home on the sixteenth post-operative day.

Comment.

Although it is far too early to make any very definite observations about this procedure, there is no doubt that this patient has been very greatly improved.

It must be emphasized that the indication for an anastomosis is generalized portal hypertension with hæmatemesis. Ascites alone is not an indication for an anastomosis. Ascites alone without other evidence of portal

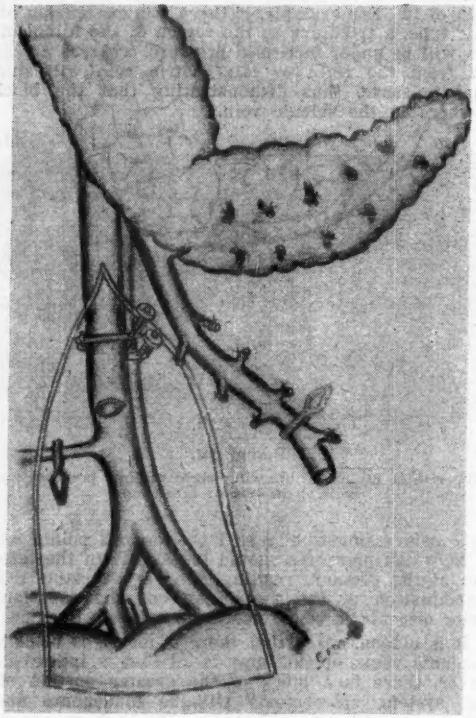


FIGURE VII.

Vessels prepared for anastomosis. Splenic vein separated from pancreas and trimmed. Renal vein incised. Clamps in position on renal vessels, splenic vein and ovarian vein.

hypertension is usually due to hypoproteinæmia resultant upon liver damage. Ascites occurring *pari passu* with portal hypertension and hæmatemesis will be greatly relieved by the anastomosis, and will be further greatly relieved by forced protein feeding.

In assessing the value of these "patch-work" procedures, one's purpose must be quite clear. Obviously, such patients can never be normal people. At best, the procedure relieves the patient of distressing symptoms and frequently considerably delays the onset of a fatal hæmatemesis. If this correct perspective is maintained, neither will extravagant claims be made nor a useful procedure be condemned.

Summary.

1. The types of portal-bed block are indicated.
2. Relevant points in diagnosis are mentioned.

3. The rationale of the operative procedure is indicated.
4. The technique of spleno-renal anastomosis is described.
5. An illustrative case is presented.

Acknowledgement.

My thanks are due to Dr. Peter Crooke for the diagrams presented in this article.

CŒLIAC DISEASE AND THE CŒLIAC SYNDROME.

By G. E. M. SCOTT,[1]
Melbourne.

IN 1946 I published a review of cases of cœliac disease. In addition to the low death rate of 8·2%, it was also shown that the outcome in patients who survived was more satisfactory than in other published series. Growth was unimpaired, weight was substantially maintained, and steatorrhœa as a legacy was rarely encountered. This result may have been due in part to mobilization of vitamin *D* mainly by mechanisms other than absorption from the bowel, and possibly depended somewhat on clinical conditions which provided for treatment in sunlight. However, the course of the disease was similar to that encountered elsewhere, and the general characteristics agreed. For example, it was confirmed that cœliac disease is not an afebrile condition, although the range of temperature rise was not high. Fibrocystic disease of the pancreas, as discussed by Anderson, was referred to, and an endeavour made to link up this condition with the problems of infantile intestinal derangement. It was emphasized that an absorptive defect alone would not explain all cases of cœliac disease, and the suggestion advanced was that the disorder might also be due in part to vitamin deficiency, and be related to a toxic infective process.

More recently considerable interest has been aroused in Anderson's work, and in fibrocystic disease of the pancreas. Farber's investigations have been widely discussed, and generally have formed, with Anderson's researches, the basis of a theory which tends to separate cœliac disease and cystic fibrosis of the pancreas as separate entities. It is unlikely that the latter is a new disease, and support is lent to this notion by the fact that careful search in the fields of biochemistry and morbid anatomy has uncovered a number of cases otherwise unsuspected, and confirmed the uniform incidence of the disorder. It is highly probable that many examples of intestinal disorders, accompanied by a syndrome of malnutrition and resulting in fatal pulmonary complications, were in reality associated with pancreatic disorder. These would be variously classified under the headings of gastro-enteritis and malnutrition, accompanied by pneumonia, or even suggestive of bronchial asthma.

As many of the cases collected by Anderson from the literature revealed other features, such as hepatic changes, in addition, it might be argued that undue emphasis has been placed on pathological changes found in the pancreas in so-called fibrocystic disease of that organ. An endeavour has been made, both by Anderson and by Farber, to evolve an hypothesis which will bring all cases under one ætiological status, and account for all the morbid findings as relative and indivisible. It may fairly be stated that such a tendency is almost universal in clinical medicine, and indeed is almost always fully justified. It is, however, dependent for success on a clear understanding and an adequate comprehension of all contributing factors, as well as of their relation and importance to each other and to the developed disease process under investigation. Fibrocystic disease of the pancreas presents unexplained features, which require further elucidation.

Sheldon rightly emphasizes that cœliac disease is not the only cause of steatorrhœa in young children. After mentioning other causes, he points out that the "clinical state" which results from fibrocystic disease mimics very closely that of cœliac disease; for steatorrhœa, malnutrition of severe grade and abdominal distension are features common to both disorders. Sheldon then goes on to comment that all the conditions he refers to have generally been designated by the title cœliac syndrome. This term he considers implies that cœliac disease is "not a separate clinical entity but a state compounded of a group of symptoms which can be broken down into various causes". Sheldon considers that by clinical investigation and laboratory aids other causes may be excluded, a residue of cases being left which exhibit the "original clinical concept of cœliac disease". At the same time he admits that cœliac disease remains an idiopathic condition, but believes that mistakes will be avoided by retention of the name, in order to avoid confusion with other causes of steatorrhœa, a confusion encouraged by using the term cœliac syndrome. Sheldon's argument is, however, circular, for the original clinical concept of cœliac disease is nothing more than steatorrhœa, malnutrition and abdominal distension. Clinical investigation and laboratory aids will not separate anything from such a clinical concept, rather will they add to the understanding of its nature. It cannot be too strongly stressed that all the factors operating in the condition known as cœliac disease or cœliac syndrome (by whatever name it is called) are not fully understood. Consequently, until all these factors are elucidated, dogmatism as to the part played by any one of them is inadmissable.

Pancreatic Lesions.

In 1942 Farber postulated two clinical pictures. The first is represented by meconium ileus and the second is a variant of the cœliac syndrome known as cystic fibrosis of the pancreas. The latter is constantly found associated with dilatation of the ducts and acini of the pancreas, inspissation of its secretion, and variable degeneration and atrophy of the parenchyma together with fibrosis. The explanation of such diverse pictures as intestinal obstruction in the newborn and the cœliac syndrome, Farber accounted for by a theory relating to the time of occurrence of the pathological changes in the pancreas. Thus, if pancreatic achylia is present during the latter part of intrauterine life, there occurs an altered state of the meconium, which results in bowel obstruction shortly after birth. When pathological changes occur after the neonatal period, the cœliac syndrome is presented. Farber undertook in collaboration what are described as "enzyme studies", with the result that three tentative divisions are postulated, namely, true cœliac disease, displaying normal enzyme values in the duodenal contents, selective enzyme deficiencies, and finally cystic or pancreatic fibrosis. In the group of selective enzyme deficiencies are included cases revealing an important diminution in any one of the three enzymes found in the duodenal juice, the other two being within normal limits. The group comprising cystic fibrosis of the pancreas present almost complete absence of lipase, amylase and trypsin—that is to say, pancreatic achylia. *Achylia pancreatica*, Farber asserts, is probably produced by "an intrapancreatic disturbance of the formation, liberation or passage into the duodenum of pancreatic enzymes". Farber and co-workers concluded from post-mortem studies that congenital atresia or stenosis of ducts was a rare cause of pancreatic achylia. Furthermore they believe that, although neither vitamin *A* deficiency nor the presence of a filterable virus plays a primary part in the pancreatic lesions or is essential to their development, either may complicate the picture. The hypothesis put forward is that the pancreatic disorder is part of a general disease, having its chief incidence in the pancreas. This disease postulate is gained by analogy from the pancreatic lesions, which reveal a more destructive form of the disorder. The widespread nature of the process is claimed to be revealed by dilated granular structures filled with inspissated material. The location of the changes is in the upper part of the respiratory and intestinal tracts, the gall-bladder, and elsewhere. Farber and colleagues therefore find support for the theory of Blackfan and Wolbach, namely, that the genesis of fibro-

[1] Armytage Medical Research Prizeman, 1949.

cystic disease of the pancreas resides in the production of an abnormal secretion by the pancreas. Further confirmation is provided, according to these workers, by demonstration that the clinical picture of the cœliac syndrome associated with pancreatic achylia may occur at a time when the only abnormal changes found in the pancreas are represented by inspissation of secretion in the acini and small ducts, and no cystic fibrosis or atrophy of cells is revealed. Experimental proof of this hypothesis was attempted by Farber, who treated kittens over a period of two to eight weeks by the subcutaneous administration of parasympathomimetic drugs, of which the most effective proved to be pilocarpine hydrochloride in doses increasing from 0·5 to 1·0 milligramme three times daily. The outcome was extreme loss of weight of the animals, together with severe nutritional disturbances. It was claimed that on histological examination of the pancreas, lesions were found similar in all important respects to those present in infants the subjects of fibrocystic disease of the pancreas. Thick homogeneous eosinophilic material filled the acini and small ducts of the pancreas. Control experiments were claimed to show that dehydration and starvation were not of primary importance in the results obtained. Farber and co-workers contemplated further investigation of the nature of the disturbance of the secreting mechanism.

In 1944 Farber reported 18 cases of fibrocystic disease of the pancreas in which post-mortem examinations had been made. Here it is now asserted that stenotic and atresic duct lesions of the pancreas are important (Landsteiner). Treatment of the material in meconium ileus, it is claimed, may be undertaken with production of a fluid state in the intestinal contents, and consequently this procedure is enjoined as a therapeutic measure as well as a diagnostic aid. Consequently a distinction is drawn between meconium ileus caused by pancreatic achylia and the state of inspissated meconium due to partial or complete obstruction of the intestine in the neo-natal period. Farber adds: ". . . there is no clinical method of determining which patient with meconium ileus will develop symptoms referable to other organs in the body and which will have the disturbance limited to the consequences of pancreatic achylia." This statement seems to beg the question at issue, for if the pancreas was not involved, according to Farber no cœliac syndrome would supervene.

In another paper, also published in 1944, Farber reported results of a study of post-mortem material from 87 cases of fibrocystic disease in infants. Changes "essentially the same as those in the pancreas" were revealed in the lungs, liver, gall-bladder, and upper parts of the alimentary and respiratory tracts. On this evidence the assertion is made that what is called fibrocystic disease of the pancreas is "really a systemic disease with a variety of clinical appearances, the occurrence of which depends on when the obstructive changes occur and which organs are affected". Pancreatic achylia then, according to Farber, is a part of this systemic disease. On the basis of this theory an investigation of the nervous control of the pancreatic secretion and of the mucous glands appeared to be a logical approach to the nature of the systemic disease process. Reference is then made to the change in the physical characteristics of mucus and to the effects of vagal stimulation on the secretion of the pancreas. This may indicate the previous experiments conducted by Farber and associates already referred to in this paper. Farber mentions the widespread involvement of mucus-secreting structures in the body, and indicates the possible importance of either a deficiency or an insufficiency of mucinase required for the maintenance of mucus in the normal physical state. The character of the duodenal secretion in fibrocystic disease, which Farber and associates found to be viscid in nature, is reminiscent, they say, of inspissated secretion in the pancreatic acini. A disturbance of parasympathetic innervation, or perhaps more accurately an "autonomic imbalance" in the nervous control of secretion in the pancreatic and mucous glands, is claimed to be suggested by the findings. Farber further believes that obstruction may commence in the individual cells of the pancreatic acini. Atrophy of acinous structures ensues, also fibrosis of the gland, and dilatation of ducts results. The mechanism postulated is that pancreatic achylia is present from the time inspissation of secretion occurs in a sufficient number of acinous structures, and a long time before these structures undergo atrophy and the ducts show dilatation. The latter results are late consequences of intrinsic obstruction. In support of these contentions Farber refers to the case of an infant, fourteen months of age, having clinical features of the cœliac syndrome and no evidence of pancreatic enzyme activity. At the post-mortem examination the gross appearance of the pancreas was normal. Microscopically no atrophy of acinous structures, fibrosis, or dilatation of larger ducts was found. However, there was dilatation of small ducts, and the acini contained inspissated secretion, which appeared effectively to obstruct the outflow of pancreatic juice.

If this case is taken, as Farber explains, to illustrate his theory of the underlying pathology of fibrocystic disease, then it may be assumed that the abnormal secretion of the pancreas, deficient in enzyme content, is prevented by blockage of a variable proportion of the acini and small ducts from entering the larger ducts and thus finding its way into the duodenum. However, it is asserted that in the case described there was no evidence of pancreatic enzyme activity. It follows, therefore, since the duodenal contents obtained were analysed, that the abnormal secretion was produced by acini which were not the seat of blockage by inspissated material, and found its way by ducts which were not blocked. No atrophy of acinous structures was found, and therefore it must be assumed that secretion produced by a certain number of normal unblocked acini was deficient in enzyme activity.

However, the fact that pancreatic acini appear normal in a pathological specimen does not prove conclusively that the secretion produced in them is normal or abnormal, for changes not revealed by methods of preparation of the specimen may have been present. Nor does the presence of inspissated material in acini and ducts necessarily imply that these structures are blocked, and that no secretion is able to escape from them. Indeed, the viscid nature of the external secretion of the pancreas described by Farber, Shwachman and Maddock strongly suggests that such material does leave the acini and ducts to enter the duodenum. Farber's description further suggests that a later stage of the process results in atrophy of acini, which implies that blockage by inspissated secretion is the cause of such atrophy. No convincing explanation is offered for the further complication of fibrosis.

If in the early stages of the disease many acini are normal and a large number of ducts patent, then, with the proviso as to interpretation of pathological preparation referred to above, it can be concluded that the pancreatic juice should reveal some enzyme activity. This, however, is stated not to be the case in cystic fibrosis. The only conclusion to be drawn is that the secretion has been altered by means of some other mechanism, and that this mechanism may be related to physiological processes. It appears that no other explanation will account both for the selective enzyme deficiencies of Farber's hypothesis and for cases of fibrocystic disease. We are thus thrown back on what appears to be the original theory put forward by Blackfan and Wolbach, namely, that the diseased pancreas produces an abnormal secretion. This altered secretion may, under circumstances at present unknown, become inspissated, but in any case it appears to be deficient in enzyme content. There is no proof that this altered secretion, together with the declared side effects, is related directly either to atrophy of pancreatic cells or to the fibrosis found present at a later stage of the morbid process. Such an hypothesis is nevertheless inherent in and implied by Farber's descriptions.

Evidence that deficiency in pancreatic juice may be a temporary occurrence is furnished by the investigations of Philipsborn and associates, who divided their patients into four groups. The first group comprised infants who were afebrile and in a good state of nutrition. The second group included those infants with feeding problems who were not maintaining adequate nutrition but were free from apparent pathological processes. Group three was

made up of patients with fibrocystic disease of the pancreas, and group four of patients with cœliac disease having the characteristics described by Gee. Duodenal intubation with fluoroscopic control was performed. Intravenous administration of secretin was used, or intraduodenal administration of decinormal hydrochloric acid. In cases of fibrocystic disease "obliteration" of pancreatic activity was found. In the group with feeding problems a state of temporary pancreatic insufficiency was revealed, and in the cases of cœliac disease normal results were obtained.

This appears to be the only investigation published in which an attempt has been made to correlate physiological activity of the pancreas with the morbid conditions included in the cœliac syndrome. As a consequence, some experimental work on the mechanism of production and composition of pancreatic secretion is apt in reference both to the work of Philipsborn and associates and to that of Farber. Harper and Raper endeavoured to discover whether secretin and vagal influences were the only factors concerned in the initiation of pancreatic secretory activity. They commenced from the accepted theory of the dual hormonal and nervous control of pancreatic secretion. Secretin from the intestinal mucosa controls the fluid and bicarbonate, and the vagus the enzyme content of the pancreatic juice. They affirmed that the question of whether or not secretin was also an enzyme stimulant was unsettled, and that a sharp demarcation of hormonal and nervous control had not been confirmed. They considered the possibility that the intestinal epithelium might contain a hormone capable of stimulating the production of enzyme by the pancreas. Preparations were made in the form of extracts from the upper three or four feet of the pig's intestine. These extracts were found to be effective in promoting an enzyme-rich pancreatic secretion on intravenous injection into cats. Pancreozymin, as the active principle was named, was found to be effective in animals who had undergone vagotomy and in those subjected to intravenous administration of atropin. Effects were obtained equally in starved and well fed animals, and the possibility was excluded of a vasodilator substance in pancreozymin which might account for the results obtained. Parallel yields of amylase and trypsinogen were obtained, similar to those produced after vagal stimulation. The work of Harper and Raper has been confirmed by Greengard, Grossman, Woolley and Ivy, accepted as being of some importance, and referred to by Kahlson in recent studies of gastric and pancreatic secretory activity.

It may be affirmed, therefore, that there is evidencec that pancreatic secretion may become temporarily deficient in enzyme content, and that, provided some concurrent intestinal disorder resolves, the secretion may again reveal the presence of ferments. Furthermore, there is some evidence that the enzyme content of the pancreatic secretion is dependent on a substance which has been named pancreozymin, and which is formed in the wall of the intestine. It may then be argued that temporary changes brought about by factors related to diet may indirectly alter the enzyme content of the pancreatic juice by inhibiting pancreozymin formation. Such factors might range from irritation of the bowel wall by unsuitable diet to mild grades of inflammatory reaction. Furthermore, in cases of gastro-enteritis such factors would be expected to be operative and possibly result in more prolonged deficiency in enzyme production.

Poynton, Armstrong and Nabarro published a contribution to the study of a group of cases of chronic recurrent diarrhœa in children. In one case reported the intestinal flora was too diverse to allow any definite conclusion as to ætiology, although changes were found in the intestinal tract from the stomach to the colon. The findings comprised congestion of vessels of the submucosa and small-cell infiltration amongst gastric glands. Cell aggregates in the form of nodules occurred. There was increase of perivascular fibrous tissue. There were fatty changes in secretory cells as well as the other changes mentioned in the duodeno-jejunal flexure. Similar changes, together with extreme congestion and hæmorrhage, were found in the colon. In the pancreas there was marked increase of interlobular fibrous tissue, especially around ducts. The appearance of certain ducts suggested desquamation of the lining epithelium. Congestion of blood vessels was revealed, and blood in the veins contained polymorphonuclear leucocytes. The gland tissue appeared "natural". The liver showed fatty degeneration, and the spleen was enlarged. The lungs, however, were "small but natural". The patient revealed during life the characteristic features of cœliac disease. This case is included by Anderson (together with the cases reported by Hess and Saphir and Clarke and Hadfield) in her series of cases of fibrocystic disease collected from the literature. Indeed, in the case which in 1938 started Anderson on her investigation, similar changes in the bowel were revealed. The intestine was moderately infiltrated with lymphocytes, plasma cells and occasional polymorphonuclear leucocytes. Fibrosis of the submucosa was also seen.

It is true to say that minimal pathological changes in the bowel wall and in the pancreas, in cases in which the features of cœliac disease are exhibited, have been either overlooked or ignored when present. Thus Parsons, in his comprehensive review, referred to "two or three cases" of cœliac disease in which "some excess of fibrous tissue or small cell infiltration around the ducts of the pancreas" occurred. He regarded such "scratching about", revealing alteration in a few cells, as insufficient to justify an "exulting cackle". This quotation is referred to by Hess and Saphir, who comment that, while such changes may seem insignificant at the time of examination, they constitute a "document of a past insult", and that they may be evidence of more severe lesions in the past. It is logical and necessary from the pathological viewpoint that minimal lesions should be observed and studied. Evidence of the value of knowledge concerning early lesions and their various phases of development is not wanting in the study of pathological processes. Tuberculosis and syphilis are two notable examples.

Indeed, the early lesions of fibrocystic disease of the pancreas have been the subject of considerable investigation. Blackfan and May reported that in 2800 necropsies there were 35 examples of well defined pathological pictures of fibrocystic disease of the pancreas, the existence of which was unsuspected during life. Five of the patients affected died soon after birth, and the remainder either failed to gain or lost weight. There were frequent diarrhœal episodes, and the patients died after developing widespread pulmonary infection. In addition to these 35 cases, however, early stages of the process in the pancreas were observed in 200 cases. The changes consisted in slight fibrosis, dilatation of acini, and inspissation of secretion. A second study appeared later, when Anderson and Hodges noted that early but well defined lesions were discoverable in newborn infants considered to be the subjects of fibrocystic disease of the pancreas. In order to find the possible incidence of the earlier lesions, Paige (in association with Anderson and Hodges) examined the results of 346 consecutive autopsies of fœtuses and stillborn babies. No typical lesions were discovered. The interpretation of early lesions made by Anderson and Hodges is, however, clouded by their reference to a case reported by Kaufman and Chamberlin. In the report of this case the claim is made that lesions depicted in photomicrographs represent early changes of the nature of fibrocystic disease of the pancreas. The reproductions, however, are certainly representative of an advanced stage of the disease. Probably Anderson and Hodges are indicating a reference by Kaufman and Chamberlin to an "abortive effort" by pancreatic cells to form acini, of which one small area of one of the sections might be regarded as an illustration. A similar microscopic picture is illustrated in the paper by Hess and Saphir, who regard similar collections of pancreatic cells as being in the nature of a syncytium and evidence of regeneration, of which further proof is provided by the presence of mitotic figures. It is evident that one of the chief difficulties encountered in elucidating or attempting to discover the history of these pancreatic changes and associating them with clinical findings resides in discovery and assessment of the importance of early lesions. Thus Farber's attempt to explain meconium ileus finds its chief difficulty in the fact that only minimal lesions of the

parenchyma of the pancreas are to be found at birth. Consequently he is obliged to admit that obstruction in the form of duct stenosis and atresia plays a more important role than was at first suspected. It is most probable that developmental defects, such as intestinal atresia, volvulus and peritoneal bands are responsible for many cases of meconium ileus, the pancreas not necessarily being involved. Such ætiological factors are referred to by Kornblith and Otani, who direct attention to the views of Torkel and of Fanconi; the former attributes inspissation of meconium to inflammatory changes of the intestinal wall, while the latter stresses interference with the secretion of the intestinal glands. On the other hand, Hurwith and Arnheim conclude that focal developmental defects of the pancreas play a part. Farber's "blockage" theory will not satisfactorily explain cases of meconium ileus. Furthermore, as has been pointed out by Philipsborn and co-workers, the total activity of the entire gastro-intestinal tract, and not the activity of one organ, such as the pancreas, determines the character of the fæces. Indeed, this question becomes of vital importance when insufficient evidence to account for changes in character of fæcal material is adduced from a study of the part played by the pancreas alone in relation to intestinal functional activity.

Associated Lesions.

The type of pathological change observed in the pancreas has been invoked in order to explain, partly by means of analogy and partly by means of known facts concerning cystic disease of various organs, the disease-processes which accompany fibrocystic disease of the pancreas. Sheldon refers to the pulmonary lesions as probably due to cystic lung disease. Not only is this an extremely rare disease, but also reports of cases of fibrocystic disease of the pancreas reveal the occurrence of pneumonia, lung abscess and bronchiectasis. Anderson and Hodges conclude that the bronchiectasis is quite unlikely to be congenital in origin; they give as an important reason that cough is sufficient to account for it, and further state that progressive signs are revealed with the lapse of time. They consider that vitamin *A* deficiency, combined with some other unknown factor, is operative. In a recent study of congenital cystic disease of the lung, Bowden concludes that the theory of secondary pulmonary inflammation as a cause is not borne out by microscopic examination.

Rauch, Litvak and Steiner assert that the intestinal and pulmonary symptoms are unrelated, and that there is no "cause and effect relationship" between them. They claim that the relationship is one of a common congenital abnormality of the lungs and pancreas. From a consideration of the embryological development of lungs and pancreas, they conclude that constriction of ducts (or bronchial passages in the case of the lungs) may produce a picture resembling both the experimentally produced obstruction and the post-mortem findings in reported cases. They point out that an "anomalous organ" is highly susceptible to noxious influences, and that purulent bronchitis and abscess formation are thus explained. Norris and Tyson undertook a detailed study of the pathogenesis of "polycystic pancreas" in an infant. They concluded that the changes corresponded with those postulated for polycystic lesions of the kidney and liver. The conclusion was reached that "progressive distortion, cystic dilatation and segmentation of the ducts occurred simultaneously with replacement fibrosis, and did not prevent the normal gross development of the pancreas".

While Farber, referring to changes in the respiratory tract, liver and salivary glands as forming a part with the pancreas in a basic disease process, seems to lean on analogy, Anderson and Hodges leave no doubt that they rely solely on analogy to explain hepatic lesions. While overlooking many reports of fatty changes in the liver appearing in the literature (including those in Anderson's original collected cases from the literature), they postulate a biliary type of hepatic cirrhosis as the associated pathological change in the liver. They refer to local areas of cirrhosis two to three centimetres in the largest dimensions. These areas histologically suggest, they say, the pancreatic lesions, because they consist of a concentration of small bile ducts containing eosinophilic concretions surrounded by fibrous tissue. The basis of the belief appears to be in reports of biliary cirrhosis associated with pancreatic fibrosis. If it is desired to establish the theory that there is but one history of pancreatitis which is not acute in onset, then it would appear to be necessary to force ætiological factors into one mould. Such reasoning may, however, be wholly untenable. Anderson and Hodges go on to say that in the cases they refer to it was not clear whether or not they were on the basis of intrahepatic and partial biliary obstruction, since none of the patients were jaundiced. To draw an analogy between bile ducts and pancreatic acini, together with their contents which represent entirely different materials, is unsafe; but in the absence of complete data of the causative factors responsible for the changes observed, such reasoning would appear to go beyond the limit of justifiable speculation. Rauch and associates appear to have summed up the position when they wrote: "Crystallization of a symptom complex diagnostic of cystic fibrosis of the pancreas cannot be said to have been perfected." So-called chronic pancreatitis has received rather scant attention in pathological writings, chiefly owing to doubtful ætiology, and there are few detailed descriptions of the disease. However, since the advent of what has been named congenital cystic fibrosis of the pancreas, many detailed descriptions of this disorder have been published, and it has been regarded as a new and separate entity on insufficient evidence. The descriptions given in recent years vary in no substantial degree from those formerly given. The gland reacts to injury in the same general way as does any other tissue. For example, atresia of the main ducts, if of sufficient degree, will result in atrophy of secreting elements and their replacement by adipose tissue. Doubtless lesser degrees of duct narrowing or intermittent pressure changes produce varying atrophy of the cells, banking up of secretion which becomes thickened, and fibrosis. But atrophy of acini may also be the result of malnutrition with resulting changes in the composition and physical state of the secretion; and fibrosis may be caused by such widespread disease as that associated with congenital syphilis. Inflammation from whatever cause may overlie other processes and be evidenced by engorgement of vessels and cellular infiltration. There is indirect evidence that pancreatic changes in the cœliac syndrome result from the incidence of malnutrition, as well as being dependent on toxic and possibly infective agents. For example, ascending infection of the duct system may result in glandular changes similar to those found in fibrocystic disease.

Toxic and Infective Factors in the Cœliac Syndrome.

The first step in establishing the toxic and infective factors in the cœliac syndrome is to dispose of meconium ileus *per se* as a factor and to discuss the possible congenital origin of fibrocystic disease. Meconium ileus, which is nothing more than an example of intestinal obstruction of a particular kind, has been closely associated with the cœliac syndrome in the literature in recent years. On account of practical difficulties with newborn infants, it is not possible as a routine to estimate the activity of the pancreatic secretion, and therefore a method of providing evidence for the presence of fibrocystic pancreas is denied. Even if it was possible to analyse the duodenal contents, it would not thereby be proved that the meconium was physically altered solely on account of the composition of the pancreatic secretion. As has been pointed out, other factors may be operative. Furthermore, only the tryptic activity of the juice could reliably be estimated, and amylase is affirmed to be absent during the first three months of life.

Pancreatic achylia before birth appears to be an anomalous postulate. Meconium reaches the ileo-caecal junction during the fourth month of intrauterine life and the rectum is filled by the fifth month (Torkel). The total quantity of meconium, some 70 to 90 grammes, is expelled during the first few days of life (De Lee). There is no evidence that pancreatic secretion plays any part in disposing of meconium, for if this was so it would be

disposed of more rapidly. Indeed, the slower appearance of normal fæcal material indicates the later functioning of pancreatic ferments and biliary mechanisms. The fact that meconium is found present with a state of blockage of the intestine does not prove that its composition is dependent on the absence or presence of pancreatic secretion. Indeed, it is more probable that relief of pressure in the intestine, by expulsion of meconium, at first promotes, or acts as a stimulus to, the initiation of pancreatic secretory activity. Thus, if there was present obstruction to the bowel at birth, this alone might inhibit the formation of pancreatic juice. As would be expected, since meconium is intended to be expelled at or shortly after birth, it would not normally be susceptible to disintegration by pancreatic juice. The composition of meconium points to this probability, for meconium consists of epithelium from the intestine, bilirubin, cholesterin crystals, stearic acid, fat, bile, lanugo, skin epithelium and sebaceous matter from swallowed *liquor amnii*. Pancreatic juice is not capable of dealing with all these substances, and it is unlikely that such a process is provided for by the organism. On the other hand, it is probable that if meconium remains in the intestine for a longer period than normal, back pressure effects, as suggested by Landsteiner, will produce changes in the pancreas. Abnormalities of the pancreatic ducts, such as narrowing of the lumen, will result in effects, if any, in the neo-natal period and afterwards, and be unrelated directly to the mechanisms associated with meconium ileus.

The reason why cystic fibrosis of the pancreas was originally regarded as of congenital or familial origin appears to rest partly on certain reports in published papers and partly on its supposed connexion with meconium ileus, which was regarded as being related in some way to pathological changes in the pancreas. Harper was the first to draw attention to a paper by Garrod and Hurtley, and to conclude that certain cases which she then reported were similar to the case reported by these investigators. The case described by Garrod and Hurtley, although giving evidence of congenital and familial origin, in no way resembled a case of fibrocystic disease as now described, and there were no signs at any stage suggestive of the cœliac syndrome. Garrod and Hurtley regarded their case as one in which there was a congenital malformation of the pancreas as a result of which no fat digestion was possible, or an anomaly of the nature of alcaptonuria or cystinuria. As the patient was alive when the case was reported, no details of the pancreatic abnormality were available. The name congenital steatorrhœa was given to the condition, and as such it was described by Garrod, Batten and Thursfield. A congenital lesion is, properly speaking, not progressive, tissue damage or abnormality having taken place by virtue of factors operative during intrauterine life. Hence, any theory propounding a progressive lesion after birth in congenital fibrocystic disease of the pancreas, as it is described, is not sound reasoning. Anderson and Hodges, after a most exhaustive search in the literature and a comprehensive study of statistical material, have concluded that their collected data are capable of an interpretation which will not admit of fibrocystic disease of the pancreas being certainly of congenital origin, although it may be an expression of an hereditary trait, requiring some unknown factor for its expression.

It is possible to accept the findings of Anderson and Hodges, and determine that the other factor or factors may be infective in origin or be related to vitamin deficiency. However, the hereditary factor is not very tangible, and the theory of infection has something to support it, particularly if the lesions are regarded as being progressive in nature.

The intestinal canal of the infant becomes infected through the mouth and the anus within twenty-four hours of birth. The normal flora of the bowel is established, and other organisms which may develop pathogenic action are acquired.

Absorptive Defects and the Cœliac Syndrome.

There is some direct evidence to show that absorptive defects alone may temporarily produce the characteristic features of the cœliac syndrome (steatorrhœa with loss of weight, wasting and prominent abdomen) in the newborn and very young without involvement of the pancreas. Examples of this condition have been furnished by Begg and Pontius, in cases in which bowel resection has been performed in infants suffering from intestinal obstruction. In one case 118 and in the other 55 centimetres of the ileum were resected. One patient died 112 days later from inanition, but after the cœliac features which had appeared had subsided. Lung infections were frequent. In the other case, in which cœliac signs also developed, the patient responded well to pancreatin, but died of a respiratory infection during a ward epidemic. The pancreas in both instances was reported as being normal. The suggestion from this evidence is that it may reasonably be postulated that malabsorption in infants may result in cœliac manifestations and be associated with a marked tendency to infection, but that this state may resolve. In this event, then, the history of the case will present features of cœliac disease, which is represented as not exhibiting irreversible tissue changes, as are revealed in fibrocystic pancreas, hepatic cirrhosis and vitamin deficiency states.

There is some evidence in support of a theory that the cœliac syndrome is a state of malnutrition initiated and maintained by an infective process in the intestine, and that at times the derangement of physiological processes in the organism may initiate and maintain tissue changes which become irreversible. These events are illustrated in the occurrence of epithelial metaplasia in the bronchial tree and in ducts such as those of the pancreas. Xerophthalmia is another example. Doubtless both these are associated with vitamin *A* deficiency. Fatty infiltration and necrosis of cells may occur in the liver. The necrosis is not zonal but results sometimes in portal cirrhosis. Glands such as the parotid and submaxillary show fatty infiltration and fibrosis. The pancreatic cells are atrophic, and fibrosis may occur. Cellular infiltration in the pancreas and salivary glands has been observed, and in the former case generally regarded as of no undue significance. On the other hand, tetany is an example of recoverable change, and rickets does not develop under certain conditions where growth can be maintained at a level sufficient to ensure absence of permanent bony changes. There is evidence of minimal pathological changes in the intestine, including the stomach, in cases regarded as examples of cœliac disease, but these changes have been ignored or regarded as of little consequence. In some cases collected from the literature by Anderson, and regarded as examples of fibrocystic disease, similar changes were revealed in a more pronounced degree. Another problem arises in connexion with associated hepatic and pancreatic lesions. It has been known since the time of the discovery of insulin that the depancreatized dog maintained on insulin develops fatty liver changes. These pathological changes were regarded as being due either to the absence of pancreatic enzymes from the bowel or to a defect in fat transport in the body. Support for the latter hypothesis was found when it was discovered that dogs fed with lecithin developed a similar process and that choline also was effective as a preventive. If rats are fed on a diet of high fat and low protein content, fatty liver changes which otherwise appear may be prevented by administration of choline.

Best, Huntsman and Redout define the term "lipotrophic" as representing substances which decrease the rate of deposition and accelerate the rate of removal of liver fat. Allan, Bowie and McLeod consider that in some way the continued absence of the pancreas will result in a pathological condition in liver cells, resulting in accumulation of fat within them. They mention that, as this change does not occur in *diabetes mellitus*, the pancreas may produce some other internal secretion necessary for physiological integration of the liver. Best and Hersley provided experimental evidence that the addition of crude and purified lecithin from egg yolk and fresh beef liver to the diet of normal white rats prevented accumulation of fat in the liver. Friedman and Friedman conducted experiments with male albino rats. They found that when a diet high in fat and low in protein content was fed to the rats, changes occurred in the pancreas and liver. The acinar tissue of the pancreas showed degenerative

changes, and fatty infiltration and degeneration were observed in the liver. More recently methionine has been regarded by Sellars *et alii* as the key substance in the sequence of these fatty changes. However, McHenry and Patterson, in a review of ætiological factors relating to fat transport, concluded: "The present situation with regard to the cause and prevention of fatty livers in depancreatized dogs is that there are a number of unanswered questions, which seem to apply equally to the human being."

An interesting study bearing on this question is reported by Dixon and associates, who studied patients who had undergone pancreatectomy for carcinoma of the pancreas. The stool fat content increased, but no diarrhœa occurred. Half of the fat ingested, as well as one-third of the protein, was lost in the stools. Absorption of calcium and phosphorus was apparently adequate. The administration of pancreatin reduced the loss of fat and protein by 50%. The patient gained weight during the period of the controlled dietary study which was undertaken. No hepatic deficiency was revealed by the sulphobromphthalein test during a period of eight months. Hyperlipæmia, rather than hypolipæmia, resulted. These workers, however, note the possibility that the changes observed in depancreatized dogs may ultimately develop.

Wollaeger and co-workers have also investigated the efficiency of the gastro-intestinal tract after resection of the head of the pancreas. Ten patients were treated surgically, two for severe chronic pancreatitis, the other eight for carcinoma, situated at the ampulla of Vater in four cases, the duodenum in one case, and the head of the pancreas in three cases. In three of the cases of involvement of the ampulla the pancreatic ducts were ligated, and in the remaining case portion of the pancreas was anastomosed to the jejunum. In one of the cases of pancreatitis the remaining portion of the pancreas was anastomosed to the stomach, and in the other to the jejunum. In three of the cases of involvement of the head of the pancreas an anastomosis was made to the jejunum. "Intake-excretion" studies were made with standard test diets. Results showed that increase in fat and protein in the diet resulted in increased loss of these substances in the fæces, but also there was an increase in the amounts assimilated. Diets taken had a high content of Calories, protein and carbohydrate and a low content of fat. Pancreatin was administered if impaired gastro-intestinal function was severe. In these circumstances most patients who were submitted to resection of the head of the pancreas maintained a satisfactory state of nutrition.

These studies of complete and partial pancreatectomy, which have been considered at some length, have a bearing on problems relating to fibrocystic pancreas. Poor absorption, particularly of protein but also of fat, has been stressed as playing a very important part in the results of this disease. The results of the investigations referred to point strongly to the necessity for a cautious attitude on the question of the relation of pancreatic disorder to nutritional states, and underline the need for consideration of other factors as being of some importance. For example, it may be suggested that the irreversible tissue changes already referred to take place only in the presence of some other factors, such as infective and related toxic states.

In reference to absorption of fat, and of carbohydrate in particular, recent work by Frazer and by Emery respectively is probably of considerable importance. Frazer's investigations suggest that fat may be absorbed in two different forms, and Emery brings forward evidence to prove that absorption of glucose in cœliac disease is not defective. If the latter finding is confirmed then glucose may be useful to spare protein, which would thus be available to provide the lipotropic factor. In regard to fat absorption Sheldon poses the question whether either one or both mechanisms of fat absorption may be deficient in cœliac disease, and Emery finds that carbohydrate metabolism corresponds to that in other marasmic states.

Pathology.

Material.

The study in this section is made from post-mortem material in the form of microscopic specimens. It relates to 16 patients treated at the Children's Hospital, Melbourne, between the years 1945 and 1948. The diagnosis of fibrocystic disease was in all cases confirmed at autopsy by microscopic examination of the pancreas. In each case pathological changes were observed sufficient in degree to establish such a diagnosis. Table I shows the sex and age at death of the sixteen patients.

TABLE I.

Case Number.	Sex of Patient.	Age of Patient at Death.	
		Years.	Months.
1	F.	4	6
2	F.	10	0
3	F.	9	0
4	M.	4	6
5	F.	1	5
6	F.	5	6
7	F.	—	4½
8	F.	—	6
9	F.	1	0
10	F.	—	6
11	F.	1	11
12	M.	—	6
13	M.	—	7
14	M.	—	8
15	F.	—	8
16	F.	—	4

It will be seen from Table I that there were twelve females and four males, whose ages ranged from four months to ten years. Only eight patients were under the age of one year; these comprised five females and three males. Of the remaining eight patients, seven were over the age of one year and one was aged one year; this group included only one male child. It will be noted that there were two patients aged nine and ten years respectively, both females. It is revealed also that female patients survived the longest, the only male survivor dying at the age of four and a half years.

Pathogenesis.

There are a number of features of these cases which are of interest.

Examination of the pancreas in Case II revealed extensive fibrosis enclosing, in parts, spaces filled with red blood corpuscles. There was considerable small round-cell infiltration. The acini were atrophic, and showed a tendency to rupture and coalesce. A similar picture is shown in Figure V (Case XIII). There were somewhat large spaces lined by epithelium varying in type from flattened to columnar. Such changes suggested metaplasia of duct epithelium possibly related to vitamin *A* deficiency. Changes found in the liver in this case are of considerable interest, for a collection of small bile ducts is revealed, and the picture of biliary cirrhosis is presented. These changes are illustrated in Figure XI. In another part of this section extensive fibrosis was revealed, and fatty changes were absent.

The pancreas in Case III had somewhat the same features as that in Case II. There was extensive fibrosis, and epithelial metaplasia in the ducts was seen. Vascular engorgement resembling that illustrated in Figure III (Case XIV) was revealed. The liver contained patchy fatty changes with fibrosis and some cellular infiltration.

The pancreas in Case IV presented areas of pancreatic cells cut off within areas of fibrosis. Vascular engorgement and some round-cell infiltration were evident.

The pancreas in Case V also had extensive fibrosis and some vascular engorgement.

In Case VII the architecture of the pancreas was lost, fibrosis was extensive and round-cell infiltration was in evidence.

Case VIII is of some interest, for there was extensive fibrosis with some small-cell infiltration in the pancreas; fatty infiltration of the gland was also present. The pathological picture found in the liver in this case is illustrated in Figure X, where extensive hæmorrhage

ILLUSTRATIONS TO THE ARTICLE BY Dr. G. E. M. SCOTT.

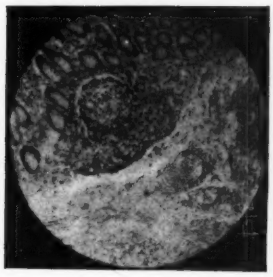


Figure I.

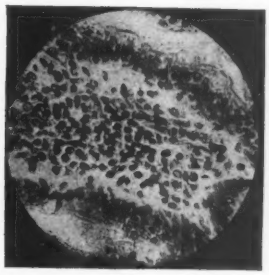
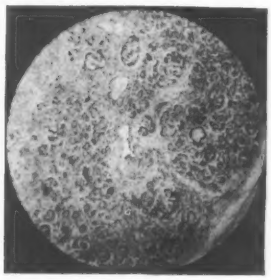


Figure II.



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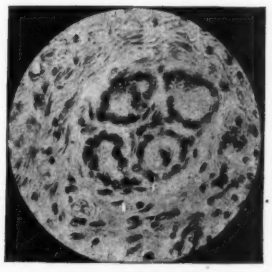


Figure IV.

Figure V.

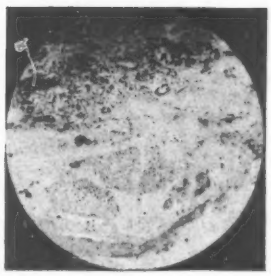
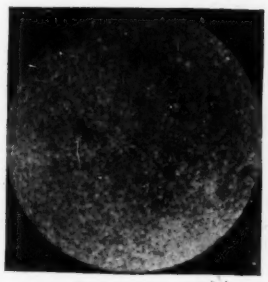


Figure VI.



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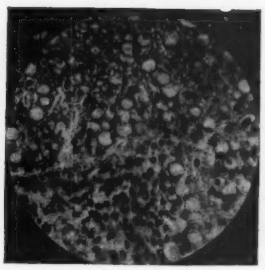


Figure VIII.

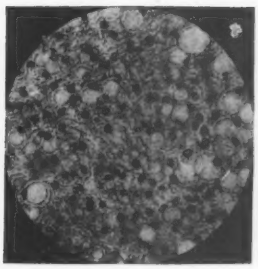


Figure IX.

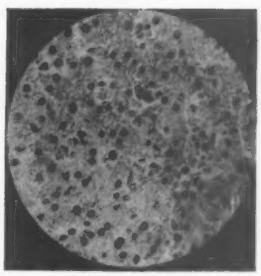


Figure X.

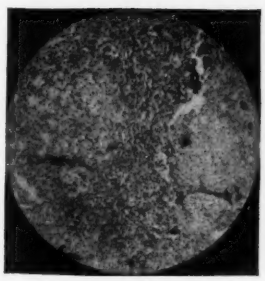


Figure XI.

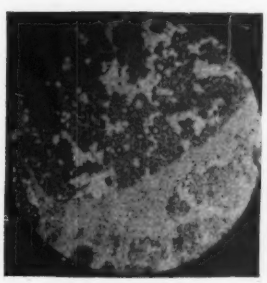


Figure XII.

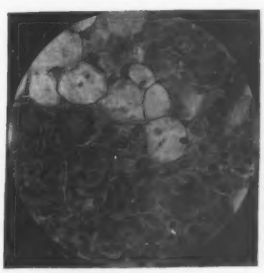


Figure XIII.

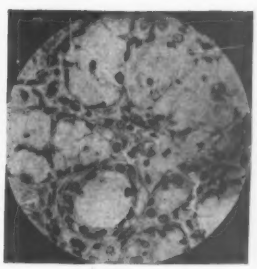


Figure XIV.

ILLUSTRATIONS TO THE ARTICLE BY DR. NOEL M. CUTHBERT.

FIGURE II.

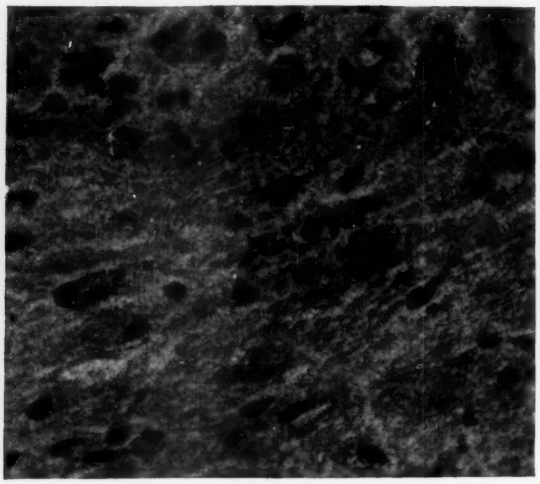


FIGURE III.

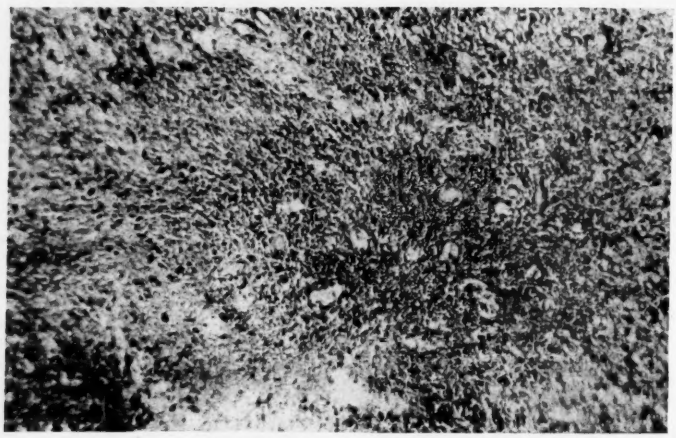


FIGURE V.

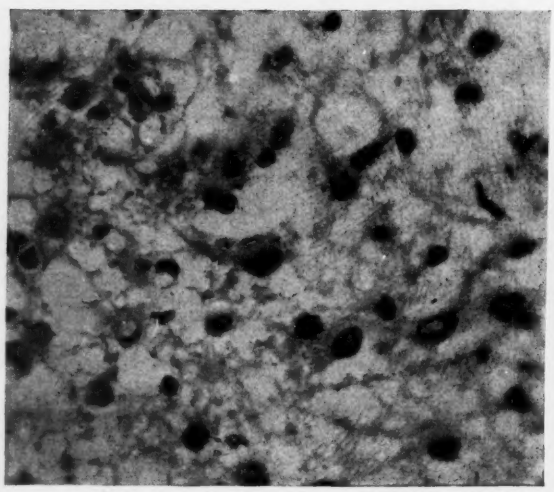


FIGURE VI.

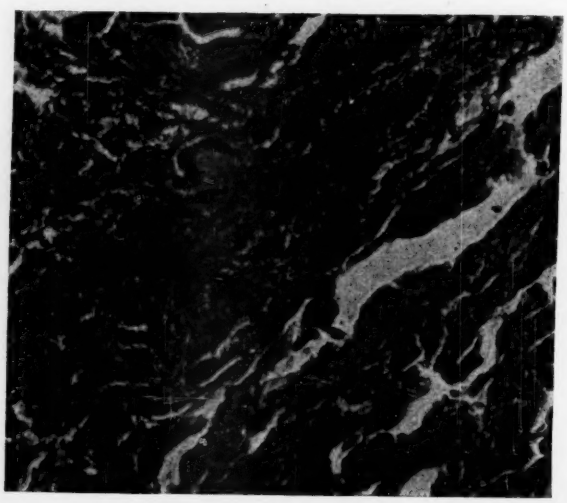


FIGURE IX.

and small round-cell infiltration are seen. No trace of hepatic cells is evident.

Metaplasia of duct epithelium was evident in the pancreas in Case X, in which a large duct revealed this change. Vascular engorgement and small round-cell infiltration appeared. The submaxillary gland in this case showed cellular infiltration.

The pancreas in Case XIII is illustrated in Figures IV and V. Here fibrosis is extensive and formed into wide bands. The acinar cells are atrophic and the acini show the tendency to rupture and coalesce already referred to (Figure V). Figure IV shows young fibroblasts between atrophic acini. The parotid and submaxillary glands in this case also contained fibrotic areas. In the parotid gland there were vascular congestion and cellular infiltration, and in the submaxillary gland cell infiltration but no congestion of the blood vessels. The fibrosis in these glands showed no tendency to assume an intralobular distribution, but was interlobular in extent.

In Case XIV examination of the pancreas, as illustrated in Figure III, reveals changes characteristic of the condition under discussion. Acini in groups are surrounded by fibrous tissue. There is cellular infiltration, with what appear to be dilated ducts. Some of these dilatations are, however, probably formed by coalescence of acini. An unusual feature is the pronounced vascular congestion. The parotid and submaxillary glands in this case were both normal.

Case XV is of considerable interest, and is represented in Figures I, II, IX, XII, XIII and XIV. Examination of the pancreas revealed what might be described as early changes, for fibrosis was seen to be minimal, and atrophy of the acinar tissue not great. The wall of the duodenum contains cellular infiltration and congestion, with fibrosis (Figures I and II). In the liver (Figure IX) are cellular infiltration, fatty infiltration and necrosis of the hepatic parenchyma. Figures XII and XIII illustrate the parotid gland in this case. There is revealed the most extensive fatty infiltration, and fibrosis is represented by a broad band in the lower part of the section. In Figure XIII it is evident that the acini are atrophic. Minimal small-cell infiltration is seen. In the submaxillary gland, illustrated in Figure XIV, the mucus-secreting elements of the gland appear normal. Cellular infiltration is fairly well marked. The submucosa of the trachea in this case contained massive cellular infiltration.

The pulmonary changes which occurred in the cases in the present series were of the nature of purulent bronchitis. No examples of cystic disease of the lung were encountered.

Much information relating to the natural history of the pathological changes of fibrocystic disease may be gleaned from a study of these specimens. One outstanding feature relates to the time interval of the development of the morbid changes in the pancreas.

Anderson, in association with De Sant-Agnese, published her views on the genesis of fibrocystic disease of the pancreas. The lesion is believed by these workers to be due to obstruction of the large and small ducts of the pancreas. The ducts of Wirsung and of Santorini may be normal, or one or both may be "atretic". Concretions in the acini and ducts may be interpreted as the result of inspissation of the secretions of the gland, and there is fibrous tissue to be found within and between the lobules. The pancreatic lesion is said to be progressive in nature, varying with the age of the patient. Thus at birth or shortly afterwards, acini are present in normal numbers and have normal structure except that the lumina of some are distended and contain eosinophilic material. Some fibrosis is present. With age the size of cysts increases, and after one year many acini have atrophied. In three or four years the acinar tissue may be entirely replaced by fat, in which a few large cysts and many islands of Langerhans remain. In all the patients who live more than a few weeks lesions are found in the respiratory tract, and these lesions are not of a nature peculiar to cystic lung disease. The pulmonary lesions are regarded as secondary to the pancreatic damage, because absent at birth, and it is further claimed that their occurrence may be prevented by means of substitution therapy and dietary measures.

Fibrocystic pancreas is said to be incident in the neo-natal period and usually to be evidenced by steatorrhœa at about the first month of life. Up to the age of six months is the period in which most of the cases are said to occur. Anderson and De Sant-Agnese claim that the life history is progressive over a period of three or four years, when most of the acinar tissue of the gland is replaced by fat. In no case in the present series was the acinar tissue entirely or even partly replaced by fat. Fatty infiltration was observed in one case (Case VIII), and there the acinar tissue was not replaced by fat. The child was aged six months at death. In the oldest patient (Case II) pancreatic changes were revealed in fibrosis of the gland. In this latter case and in Case XIII the same incidence of fibrosis occurred, and in both the acini showed evidence of rupture and coalescence, probably in the process of forming the large spaces which are described as "cysts" and frequently resemble, and may be mistaken for, ducts. The age of the patient in Case XIII was seven months. Thus the evidence points rather to atrophy of acini as occurring both early and late in the morbid process. According to the theory of Anderson and De Sant-Agnese, three to four years is the time during which acinar tissue atrophies and is replaced by fatty tissue, whereas in the present series the span is ten years at least.

Case VI, in which the patient was aged five and a half years at death, is most instructive. Here it will be seen from Figure VI that fully developed fibrocystic pancreatic changes are incident alongside normal pancreatic tissue. No theory dependent on a progressive basis for its establishment can explain such morbid findings. The pathology of this case is not clarified either by the theory of Anderson or by Farber's hypothesis. This case emphasizes the postulate that no one cause will account for all cases of fibrocystic pancreas. The pathological histology of the case might possibly be explained by supposing that stenosis of the duct or ducts of the affected lobules had been present, or perhaps by invoking the theory of an agenesis relating to the *Anlagen* of the affected part of the gland. Ascending infection of the duct system of the gland would in this case hardly be tenable, and consequently some developmental abnormality would seem to be the most likely explanation.

In all the cases in the present series, with the possible exception of Case VI, which is probably unique, the pancreatic changes differ in no substantial way from those of the morbid anatomy of chronic pancreatitis. Epithelial metaplasia in the duct system of the gland is concerned only as a complication, which doubtless is due to vitamin *A* deficiency. Cystic changes are, as illustrated in Figure V, probably due to a combination of atrophy of the acinar tissue and coalescence of adjoining acini, and not to distension of acini by retained secretion. In Figure IV it is seen that intralobular fibrosis is in the process of taking place, while the acini are undergoing atrophy. Well-formed fibrous tissue surrounds the groups of acini in both Figure IV and Figure V. The characteristic feature of all specimens of well-developed fibrocystic disease of the pancreas, the feature which makes such microscopic slides identifiable almost at a glance, is the varying sequence of large and small spaces surrounded in irregular fashion by strands and bands of fibrous tissue, interspersed by varying grades of secretory-cell atrophy. The formation of this composite picture appears to be made up in the way here suggested. Such a history of the morbid anatomy incident in the gland does not suggest congenital cystic characteristics. The presence of cellular infiltration and vascular congestion, together with fibrosis, which is revealed in the present series, does not allow the factor of infection to be ignored. Furthermore, atrophy of acinar parenchymal tissue may be due to nutritional defects in the gland, which are related to the general state of malnutrition of the patients. In material obtained by Waterlow in studies of malnutrition, examination of the pancreatic acinar tissue revealed the same atrophic changes as appear in fibrocystic pancreas. Photomicrographs illustrating Waterlow's findings show pancreatic acini indistinguishable from those

seen in fibrocystic disease of that organ, the only differentiating feature being the presence of fibrosis in the latter case. Thus there is evidence that in the two disorders, namely, Waterlow's cases of malnutrition in infants in the West Indies, and fibrocystic disease, both of which reveal evidence of malnutrition, changes in the parenchyma of the pancreas are similar.

It is therefore suggested that fibrocystic pancreas is in no way specific and hardly merits the designation of a separate disease entity, being dependent for its genesis on a variety of ætiological factors, which may vary from duct stenosis of congenital origin to infection, malnutrition and vitamin deficiency. And in addition there is no progressive march of events in the process of development of the morbid anatomical changes observed.

In regard to the hypothesis of Anderson and De Sant-Agnese associating pancreatic and hepatic changes, these workers extend their theory of abnormal pancreatic secretion to the liver, where also eosinophilic concretions in bile ducts occur, and focal areas of cirrhosis are said to be not uncommon. There is a concentration of small bile ducts which are surrounded by fibrous tissue. Atresia of the gall-bladder occurs, due to obstruction of the cystic duct consequent upon the inspissation and abnormal secretion. If, however, the changes in pancreas and liver are to be strictly comparable, then the bile-duct epithelium, which it is assumed is regarded as analogous to the acinar cells of the pancreas, would show signs of atrophy, but this is not stated to obtain.

The rare occurrence of biliary cirrhosis in childhood is of considerable interest. Infection rather than obstruction of the biliary passages is the rule. Webster has reported one very instructive case in which no obstructive lesion was found *post mortem*, but evidence of infection in a bile duct was discovered after careful search. The patient was never jaundiced. Webster believes that following infection inflammatory reaction takes place within and without the smaller bile ducts. Hepatic-cell damage of varying degree results at the periphery of the lobules and is restricted to this area. Smaller bile ducts become distended with inflammatory products, and are thus stretched. Elongation and tortuosity result. Later, owing to contraction of fibrous tissue, this phase is promoted. A section in any plane may then give the appearance of increase in the number of ducts. Figure XI (Case II) suggests an early phase of such a process, occurring at a stage before contraction of fibrous tissue at the periphery of a lobule has occurred.

Questions relating to fatty degeneration and fatty infiltration of the liver have been a perennial source of investigation and debate. Dible and Davie argue that all forms of fatty degeneration are examples of fatty infiltration of extracellular origin. If absorption of fat and protein are defective in the cœliac syndrome, then the source of this extracellular fat may be partly endogenous. It is notable that starvation may be a direct cause of fat accumulation in the liver. This possibility emphasizes the necessity of considering toxic and infective factors in the cœliac syndrome. Support for the concept of infection, toxæmia and malnutrition is derived from a recent report of cases by Perry of hepatitis due to *Salmonella derby* enteritis in children.

Hepatic necrosis of varying grades with fatty infiltration is revealed in the material here presented.

Figure VII (Case VI) indicates that necrosis is not of zonal distribution. Figure VIII from the same case illustrates marked non-granular small-cell and fatty infiltration, with a severe grade of necrosis.

Figure IX (Case XV) reveals similar changes.

Figure X (Case VIII) is illustrative of complete hepatic necrosis with hæmorrhage and minimal round-cell infiltration. It suggests severe toxæmia, possibly terminal.

Study of this material therefore suggests definite characteristics of the hepatic lesions. These are, however, probably non-specific.

Discussion.

The concept that further study and investigation will not result in splitting-off or separation of new entities or disorders from the cœliac syndrome is an important one. Further investigation will add to an understanding of the nature of the cœliac syndrome, and of the varying significance of pathological changes to be observed in association with it. For example, one of the associated factors of the syndrome was described by Ryle, who observed the association of fatty stools with obstruction of the lacteals. Patients having fibrocystic disease of the pancreas reveal signs of the cœliac syndrome, not solely or only on account of pancreatic lesions, but because other factors related to this syndrome, such as infection and malnutrition, have occurred concurrently. For example, when minimal pathological changes only are observed in the pancreas, it becomes difficult to account for all abnormalities on the basis only of the findings in the pancreas. If, therefore, the complete history of the changes brought about by pancreatic disorder in the cœliac syndrome is to be discovered, it would seem that some hypothesis must be advanced to account for deficiency of pancreatic enzymes when pathological changes observed do not reveal commensurate abnormality. In such a situation it seems reasonable to postulate an upset of physiological mechanisms brought about by gastro-intestinal disorders. Evidence has been brought to show that definite changes are found in the intestinal canal, in cases both of cœliac disease and of fibrocystic disease of the pancreas, and that gastro-intestinal disorders, which are a feature of cœliac disease, may result in pancreatic enzyme deficiencies.

This problem is well illustrated in the present series. The pancreas in Case XV had minimal pathological changes, whereas examination of the liver revealed advanced hepatitis and fatty infiltration. The parotid gland was infiltrated with fat cells and the acini were atrophic. The serous-secretory cells of the submaxillary gland were atrophic, but the mucus-secreting gland appeared normal. Incidentally, this latter observation presents evidence against Farber's theory that mucus-secreting glands are affected as part of a basic disease process in fibrocystic disease. Now if this case is compared with Case XIV, it will be observed that in the latter instance the pancreas has well-developed typical features of fibrocystic disease, whereas the parotid and submaxillary glands are normal. The importance of Case XV lies in the fact that evidence of inflammatory changes was present in the duodenum. From the point of view of its pathology the case might be regarded as one of enteritis complicated by toxic infective hepatitis and minimal pancreatic lesions, and the affection of the salivary glands regarded as secondary in the disease process. Doubtless the diagnosis of gastro-enteritis would be suggested clinically but for the accompanying features of the cœliac syndrome. At a time before Anderson conducted her investigations which focused attention directly on the pancreas, this case would certainly have been classified as one of cœliac disease. With Case XIV the emphasis is different, for pancreatic abnormality is well marked while the salivary glands are unaffected.

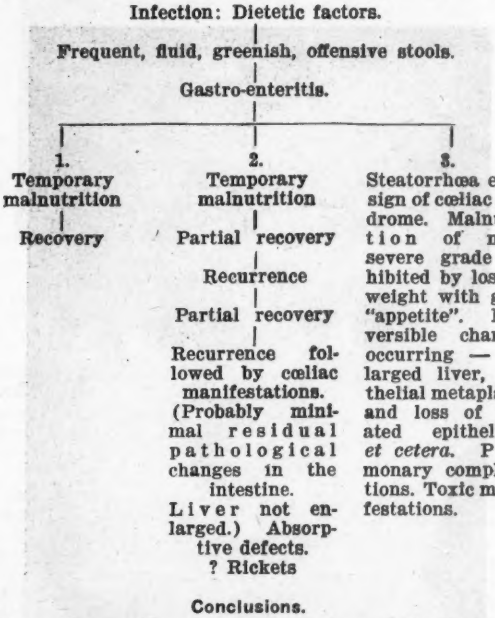
The hepatic changes in Case XV readily suggest a non-specific disorder, but such a description applied to the pancreatic findings in this case would, in the light of recent research, tend to raise serious doubts. Yet the postulate that both pancreatic and hepatic changes are of a non-specific character is not difficult to imagine. Pancreatic fibrosis and acinar atrophy may follow duct atresia, ascending duct infection, or various infective states; and hepatitis may be the result of infective and toxic factors as well as virus infections. The problem is greatly simplified if the cœliac syndrome is regarded as a physical state brought about by a number of contributing causes. The occurrence of pathological abnormalities will vary from one instance to another, and they will be more evident sometimes in one organ and sometimes in another.

When Anderson, searching the literature, in effect renamed certain cases of cœliac disease as fibrocystic disease of the pancreas on the evidence of post-mortem findings, attention was thereby directed solely to the pancreas, and other pathological findings in the cases were overlooked. When duodenal enzyme analyses were made and it was found that those patients having enzyme deficiencies were also the subjects of fatal pulmonary

disease, it was concluded that the pancreatic defect was responsible, either directly or indirectly. For instance, malabsorption of fat due to deficiency of lipase consequent on pancreatic disorder indicated deficiency of vitamin *A*, bronchial epithelial metaplasia, absence of ciliary action, and consequently increased liability to lung infections. Now while all this may be true, it may not represent the full story. It does not, for example, account for the invariable presence of the *Staphylococcus aureus* as a constant infecting agent in the lung lesions. The error of regarding the pancreas as the origin of the process lies in the resulting neglect of other possible factors, such as infective and toxic states, with resulting malnutrition. Intestinal disorder tends to be looked at solely from the point of view of the pancreatic disability, but if hepatic changes are also in evidence, storage of vitamin *A* may be disturbed, and this factor can then play a part in the developed disease process. Further, in the therapeutic field measures could be instituted in order to prevent the incidence of hepatitis. Anderson claims that early dietary measures in fibrocystic disease will prevent the onset of pulmonary complications. However, therapy in lung complications is not very satisfactory. Further information on the genesis of these changes is needed.

Poynton, Armstrong and Nabarro concluded that cœliac disease might be what was grafted on the original illness. Early in the course of some of their cases the well recognized signs were absent, for at times the patients had green-coloured or blood-stained fæces and not the bulky, pale, "porridgy" stools described by Gee. They ask: "Does the cœliac affection represent a disease in itself?" They question whether cœliac disease may not be the expression of some peculiar intestinal fault which complicates various abdominal affections, or be the result of a particular lesion arising in connexion with abdominal disease. The question of unsuitable diet is postulated, in relation to "a disease which itself still persists".

An hypothesis on modern lines may thus be illustrated.


Infection: Dietetic factors.
Frequent, fluid, greenish, offensive stools.
Gastro-enteritis.
1.
Temporary malnutrition
Recovery
2.
Temporary malnutrition
Partial recovery
Recurrence
Partial recovery
Recurrence followed by cœliac manifestations. (Probably minimal residual pathological changes in the intestine. Liver not enlarged.) Absorptive defects.
? Rickets
3.
Steatorrhœa early sign of cœliac syndrome. Malnutrition of more severe grade exhibited by loss of weight with good "appetite". Irreversible changes occurring — enlarged liver, epithelial metaplasia and loss of ciliated epithelium *et cetera*. Pulmonary complications. Toxic manifestations.

Conclusions.

The cœliac syndrome presents features indicative of a state of malnutrition, which is initiated and maintained by gastro-intestinal disorders related to infective and toxic states. For reasons at present obscure, enteritis in infancy and childhood from whatever cause may result in minimal pathological lesions in the wall of the bowel, the result of which may be reflected in a prolonged state of malnutrition, which may completely resolve. On the other hand, for unknown reasons, a state arises whereby irreversible tissue changes are produced in various structures. The outcome may be skeletal abnormalities, or may be represented by organic disease of a severity sufficient to result in death of the patient.

Summary.

1. Material obtained *post mortem* in 16 cases diagnosed as fibrocystic disease of the pancreas is examined and assessed.

2. In accordance with other published series most cases occurred in females.

3. Theories of Anderson, Farber and others relating to ætiology of the disease are critically examined.

4. Problems referring to physiology in the pancreas and liver are discussed.

5. A theory is evolved to explain the genesis of lesions found in the pancreas.

6. The pathology of lesions found in the liver is discussed.

7. An attempt is made to assess the relationship of infective and toxic states, as well as vitamin deficiency, to the cœliac syndrome.

Acknowledgement.

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Legends to Illustrations.

FIGURE I.—Case XV. Low-power view of duodenum to show cellular infiltration and fibrosis in the submucosa with evidence of vascular congestion. Hæmatoxylin and eosin.

FIGURE II.—Case XV. High-power view of duodenum to illustrate cellular infiltration by small round cells. Hæmatoxylin and eosin.

FIGURE III.—Case XIV. Low-power view of pancreas to show groups of acini surrounded by fibrous tissue and extreme dilatation and engorgement of a vessel in the centre of the section. Hæmatoxylin and eosin.

FIGURE IV.—Case XIII. High-power view of pancreas to show atrophied acini with intralobular and interlobular fibrosis, atrophy of pancreatic cells, and retained material, possibly secretion. Hæmatoxylin and eosin.

FIGURE V.—Case XIII. High-power view of pancreas to show two adjacent acini with atrophy of cells and coalescence; there is marked fibrosis. Hæmatoxylin and eosin.

FIGURE VI.—Case VI. Low-power view of pancreas to show typical fibrocystic changes adjacent to normal pancreatic tissue. Hæmatoxylin and eosin.

FIGURE VII.—Case VI. Low-power view of liver to illustrate uniform distribution of fatty changes. Hæmatoxylin and eosin.

FIGURE VIII.—Case VI. High-power view of liver to show fatty and cellular infiltration with necrosis of liver cells. Hæmatoxylin and eosin.

FIGURE IX.—Case XV. High-power view of liver to show fatty and cellular infiltration with necrosis of liver cells. Hæmatoxylin and eosin.

FIGURE X.—Case VIII. High-power view of liver to show cellular infiltration and hæmorrhage; no hepatic cells are visible. Hæmatoxylin and eosin.

FIGURE XI.—Case II. Low-power view of liver to show a collection of small bile ducts, flanked by normal hepatic parenchyma. Hæmatoxylin and eosin.

FIGURE XII.—Case XV. Low-power view of parotid gland to show extensive fatty infiltration with marked fibrosis. Hæmatoxylin and eosin.

FIGURE XIII.—Case XV. High-power view of parotid gland to show infiltration by large fat cells with atrophy of secretory elements. Hæmatoxylin and eosin.

FIGURE XIV.—Case XV. High-power view of submaxillary gland to show normal mucus-secreting elements. Hæmatoxylin and eosin.

Reports of Cases.

INTRANASAL GLIOMATA.

By NOEL M. CUTHBERT,
Perth.

INTRANASAL GLIOMATA are formed by a nipping off within the nasal cavity of portion of the anterior end of the neural tube. Nasal meningoceles or meningo-encephaloceles occur when there is a dehiscence in the cribriform-

FIGURE I.

Case I. Baby D. at age of two years and nine months (August, 1948). Note swelling on ascending process of right maxilla.

plate area. A true encephalocele generally appears in the neighbourhood of the fronto-nasal process, but, of course, can be found anywhere in the nasal region.

The tumours in the three cases described here are composed almost entirely of derivatives of medullary epithelium. They must be considered as gliomata of the astrocytoma type as the tissue has differentiated into astrocytic neuroglial tissue. Mallory's stain clinches the diagnosis in all three cases.

Nutritional deficiencies or other noxious factors in the mother affect the rapidly dividing fœtal cells, which are susceptible during certain "critical periods" of the development of the embryo. It is probable that these tumours form at this time.

Intranasal gliomata do not tend to proliferate. They are benign tumours forming no metastases. If completely removed they should not tend to recur.

The time of removal should, I think, be dependent on the severity of the obstructive signs and the possibility

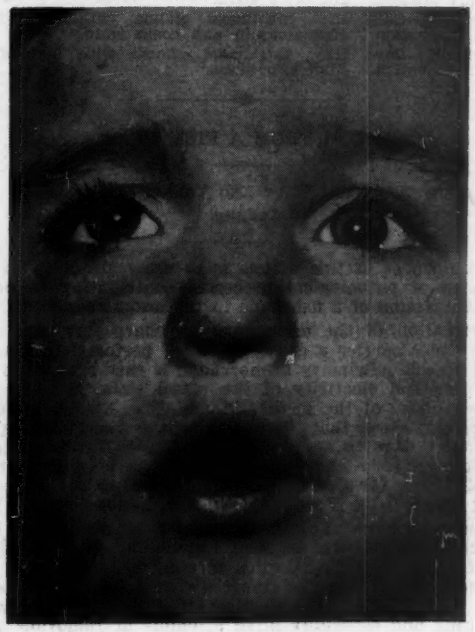

FIGURE IV.

Case II. Baby E.F. at age of one year and seven months (August, 1948). Note swelling of ascending process of right maxilla.

of resulting deformity due to pressure on adjacent bony structures.

I can find no record of intranasal gliomata having been reported in medical literature in Australasia.

Case I.

At the request of an obstetrician on November 9, 1945, I examined Baby D., then five days old. The right nostril was completely occluded by a polypus-like tumour, which was readily visible at the right anterior naris. I decided that it would be wise to defer interference and observe the patient with a view to removing the tumour when the infant was older.

On February 21, 1946, the tumour was larger and there was considerable interference with breathing, especially during feeding. Surgery was therefore indicated.

The tumour had a soft polypus-like cortex and from its centre clear fluid escaped during removal. Its base was sessile and the attachment appeared to be from the cribriform plate, the upper part of the medial surface of the middle turbinate bone, and the upper part of the septum. Adequate ventilation of the right side of the nose was thus obtained immediately.

On September 19, 1946, a further removal of tumour tissue was effected right up to the cribriform plate, as the nasal obstruction, after having been relieved, had gradually increased. The ascending process of the right maxilla was noted to be distended outwards.

Radiotherapy was considered inadvisable.

On several occasions since the first removal, the mother has stated that clear fluid ran out of the nose. This, of course, may have been a simple rhinorrhœa.

The child is now two years and nine months old and in excellent health. The nasal airway is adequate. The tissue on the area from which the tumour originated varies in size. Respiratory infections cause it to enlarge, but generally there is a tendency to regression. There is, I believe, the same tendency with meningoceles and other similar developmental lesions.

FIGURE VII.

Case III. Baby H. at age of eight months (August, 1948). Note the abnormally wide bridge of the nose, the scar on the site of removal of an extranasal tumour shortly after birth and the swelling of the ascending process of the right maxilla.

A problem lies ahead in regard to the future ventilation of the anterior group of cells of the accessory nasal sinuses.

Sections (Figures II and III) of the growth are reported on as follows:

> Neuroglial tissue is widely distributed throughout the section. Astrocytes can be found in irregular distribution. The neuroglial tissue extends directly underneath the mucosa. The density of the neuroglial fibres varies considerably. In some instances there are slightly wavy bundles, in others a more loose arrangement.

Case II.

E.F., a female child, had been noticed since her birth to have a swelling inside the right nostril.

On March 17, 1946, when the child was aged ten weeks, I removed a section from what appeared to be a hardened, epithelialized exterior of a polypus at the right naris. The pathologist's report stated that it was benign squamous cell papilloma.

On March 31, 1946, when the child was aged three months, I was able to enter the right nostril surgically

and removed two types of the tissue constituting the tumour. The first portion (the cortical) was, as I have said, similar to an epithelialized surface of a polypus. The second specimen was taken from the remainder of the tumour lying in the upper half of the nose. This was more soft and polypoid in consistency, and fluid escaped from its substance. It was identical macroscopically with the intranasal glioma of Case I, except that the attachment was not from the cribriform plate area, but from the superior meatal area laterally.

On July 6, 1948, no sign of the tumour was visible at all in the right nostril. The right nasal cavity had a cave-like atrophic look due to the previous pressure of the tumour. The prominence of the ascending process of the right maxilla is accentuated as a result.

FIGURE VIII.

Same subject as Figure VII. The scar and swelling on the right side of the nose (due to intranasal glioma) are more pronounced.

The report on the section (Figures V and VI) is as follows:

> Dense glial fibres are found in the submucosa, touching the epithelial lining here and there. Many large and small astrocytes are seen. Some of these are arranged in foci of vacuolic large cells. The neuroglial fibres are very conspicuous, especially with Mallory's stain. Some are in bundles, others are loosely distributed.

Case III.

Baby H. was referred to me at the age of two months on account of an obstruction in the right nostril which had been present since birth. When the child was aged three weeks a tumour had been removed from the bridge of the nose, but no section was taken for microscopic examination.

Examination revealed a polypus-like tumour almost filling the right nostril. It appeared to arise from the roof of the nasal cavity.

On July 27, 1948, when the child was aged seven months, I removed a small snipping for examination. The report on the section (Figure IX) is as follows:

> This picture shows a very small area of neuroglial tissue with conspicuous fibres and protoplasmic astrocytes. The fibres are very prominent when stained by Mallory's method.

The tumour will be removed in Case III when the infant is older. I am sure that a section then taken nearer the cribriform plate will be similar macroscopically and microscopically to those in Cases I and II.

I think this is a case of a mixed intranasal and extranasal glioma, as I believe the extranasal growth would have proved to be gliomatous had it been examined.

Acknowledgement.

The descriptions of the microscopic sections are by Dr. T. R. Lubbe, pathologist to the Royal Perth Hospital.

Legends to Illustrations.

FIGURE II.—Case I. Intranasal glioma with low-power magnification. Mallory's stain.

FIGURE III.—Case I. Intranasal glioma with high-power magnification.

FIGURE V.—Case II. Intranasal glioma with low-power magnification. Mallory's stain.

FIGURE VI.—Case II. Intranasal glioma with high-power magnification. Hæmatoxylin and eosin stain.

FIGURE IX.—Case III. Intranasal glioma with high-power magnification. Mallory's stain.

DEATH FROM A FISH BONE.

By ASHLEIGH DAVY AND VOLNEY BULTEAU,
Sydney.

THE purpose of this article is to draw attention to the sequence of pathological processes contributing to death from impaction of a fish bone in the œsophagus.

Perforation of the œsophagus by sharp foreign bodies at all times carries a grave threat of perforation of large blood vessels. Primary hæmorrhage is rare because of the thickness and elasticity of the vessel walls and the connective tissue of the mediastinum. Case histories almost invariably show that the hæmorrhage has taken place from four days to several weeks after the swallowing of the foreign body and that infection has been its demonstrable cause (Beal, 1917; Ter-Oganesjan, 1934; McGibbon, 1935; Clerf, 1940; Turner, 1946; Norris, 1948).

The mechanism of this secondary hæmorrhage has been described as follows: Krause (quoted by Ter-Oganesjan) is of the opinion that the foreign body damages the adventitia or even the media of neighbouring vessels. This is aggravated by pulsation of the vessel, swallowing, coughing and especially blind attempts at removal. As a result a perivascular hæmatoma is produced which then becomes infected. The infective process spreads deeper, the vessel wall is perforated and a fatal hæmorrhage ensues. In most cases there is an initial secondary hæmorrhage, which is followed by a further fatal hæmorrhage about twenty-four hours later. This is explained as being due to the fact that a thrombus brings the initial hæmorrhage to a standstill until it, in its turn, becomes involved in the inflammatory process; another hæmorrhage then starts and it is fatal. Ter-Oganesjan (1934) stresses the fact that this period between the initial and second secondary hæmorrhages must be utilized for operation.

The following case is reported because the findings at necropsy differ from those hitherto reported in the available literature.

Clinical Record.

On March 3, 1949, Miss A.A.T., aged forty-three years, was eating fish when she noticed a pain in her chest going through to her back. The pain persisted and was noticeably worse on swallowing, so that she finally consulted her doctor two days later. He arranged her admission to the Royal Prince Alfred Hospital.

At œsophagoscopy an hour or so after her admission to hospital a small piece of bone was located on the posterior œsophageal wall, nine and a half inches from the upper incisor teeth. Only about one-sixteenth of an inch of the bone was visible. On removal it was seen to be a fish bone one inch in length, and it was apparent that

it had perforated the œsophageal wall. It was firm, fine and sharply pointed. Chemotherapy with penicillin given by intramuscular injection was immediately started.

For the next few days progress was uneventful. One week after operation the patient complained that the pain had returned. An X-ray picture of the chest on March 14 was reported as revealing no abnormality. Again there was a remission of symptoms until March 19, when the pain recurred and a cough commenced.

On March 21 X-ray examination after a barium bolus revealed irregularity with obstruction in the mid-œsophageal region, some of the bolus remaining in the proximal segment after one hour. The lung fields were reported clear.

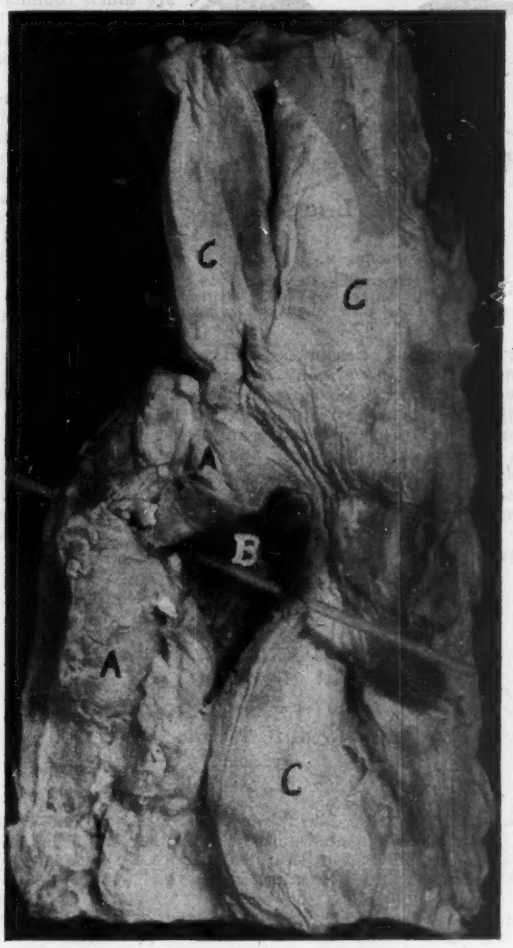

FIGURE I.

A = aorta; *B* = aneurysmal sac; *C* = œsophagus laid open.

On March 22 dysphagia was great. Some respiratory distress, cough and slight cyanosis were present. Examination of the chest revealed generalized rhonchi with diminished breath sounds over the upper lobe of the left lung, temporarily disappearing completely, and thus indicating intermittent complete blockage of the left upper lobe bronchus. It was suggested by the consulting physician that a second foreign body might be present in the left main bronchus. Œsophagoscopy was performed and the lumen of the œsophagus was found to be almost occluded by a swelling of the postero-lateral wall on the left side, in the region from which the bone had been removed. The œsophageal wall was intact. Bronchoscopy revealed that the trachea was being pressed on the left side by some external swelling, and that the orifice of the left upper lobe bronchus was almost completely occluded. A mucous plug was seen to complete the occlusion and then suddenly to permit air entry.

The findings were suggestive of a localized mediastinal abscess, but aortic aneurysm was considered possible, though no pulsation of the tumour could be detected.

A rubber feeding tube was inserted through the œsophageal constriction and the upper end brought out through the nose. Penicillin therapy was continued, and streptomycin was given, sulphonamide was given intravenously and continuous oxygen therapy was commenced.

The next morning, March 23 and twenty days after swallowing the fish bone, the patient said that she felt better. Nevertheless, shortly afterwards, she sat up in bed, had a profuse hæmatemesis and died incontinently.

At autopsy it was found that an aortic aneurysm was pressing on the œsophagus, the lower end of the trachea and the left main bronchus. It was about five centimetres in diameter and contained laminated blood clot. On examination it proved to be a "false" aneurysm. A pin-point perforation led from the aorta into the aneurysm on one hand and a ragged tear from the aneurysm into the œsophagus on the other.

Comment.

The outstanding features about this case which cause it to differ from other reported cases are as follows: (i) the presence of a false aneurysm; (ii) the absence of infection in the mediastinum; (iii) the pin-point perforation of the aorta; (iv) the larger perforation of the œsophagus; (v) the absence of an interval between the initial and the second secondary hæmorrhages.

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PREGNANCY IN A BICORNUATE UTERUS.

By PHILIP C. THOMAS, M.R.C.O.G.,
Perth.

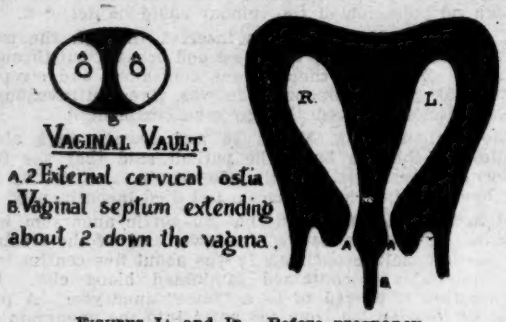
Clinical Record.

MRS. H.R., a married woman, aged twenty-three years, was first attended in January, 1948, at the time of the miscarriage at three months of her first pregnancy. At this time it was noticed that a septum about two inches long was present in the vaginal vault separating two external cervical ostia. There was only a slight fundal depression of the body of the uterus on bimanual examination *per vaginam* (Figures IA and IB).

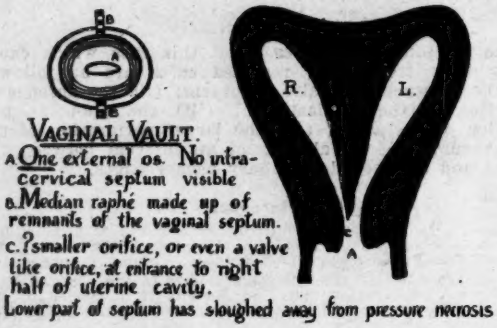
She became pregnant again the following month. The uterine condition had been regarded as some form of duplication, either bicornuate or septate, and it was thought that she was now pregnant in the left half of the uterine cavity. The ensuing antenatal progress was perfectly normal, and as there was no evidence of even potential obstruction in the pelvis she was given a trial of labour, which she negotiated successfully, delivering herself of a six-pound baby after eleven hours. Seven

weeks after the confinement examination of the vaginal vault revealed only one external os, and a median raphe represented the remnants of the vaginal septum (Figures IIA and IIB).

Hysterography was then carried out (January 13, 1949) in the ordinary way without anæsthesia; but when the films were examined it was found that only one side (the



FIGURES IA and IB. Before pregnancy.


VAGINAL VAULT.
A One external os. No intracervical septum visible
B Median raphé made up of remnants of the vaginal septum.
C. ?smaller orifice, or even a valve like orifice, at entrance to right half of uterine cavity.
Lower part of septum has sloughed away from pressure necrosis
R.
L.

FIGURES IIA and IIB. Seven weeks after parturition.

left) had filled. This gave the appearance of a unicornuate uterus, and such was reported (Figure III).

It was then decided to explore the cervical canal under anæsthesia, to seek an orifice leading off to the right. This was done on February 10; such an orifice was found, a cannula was inserted, and hysterography was repeated.

FIGURE III.
First hysterogram, January 13, 1949. Only the left side has filled. This gave the appearance of a unicornuate uterus and was reported as such.

FIGURE IV.
Second hysterogram, February 10, 1949. The right half of the uterine cavity is now defined.

This defined the right half of the cavity (Figure IV). The cannula was then withdrawn from the right orifice and quickly inserted into the left orifice, more dye was instilled and a film was taken (Figure V). Both halves of the uterine cavity were now displayed.

In the light of these hysterograms the state of affairs prior to pregnancy was probably as shown in Figure IA. Parturition itself then caused pressure necrosis and sloughing of the lower part of the utero-vaginal septum, the condition represented by Figure IIA being left.

Comment.

In the absence of symptoms and of complications in pregnancy and labour no active treatment is called for. Had the patient been examined for the first time after parturition the condition would have gone unnoticed. This bears out the often-made statement that minor degrees at least of this type of uterine abnormality are much commoner than is generally believed and, furthermore, that they may also be the cause of some of the otherwise inexplicable complications that are sometimes encountered. For example, menstruation from the non-pregnant half of the uterus is possible during pregnancy. Moreover, demonstrating that ovulation can occur during pregnancy, instances have been reported in which a woman, already pregnant in one horn of a bicornuate uterus, has later become pregnant in the other half. That is to say, superfœtation may occur in such a uterus. In theory at least many other possibilities exist for untoward happenings, and further knowledge will reveal how often the theory becomes fact. This particular case demonstrates how easily such an abnormality may remain unsuspected, and suggests that there is a place for hysterography in the investigation of certain obscure atypical gynæcological syndromes.

FIGURE V.
Second hysterogram, February 10, 1949. Both halves of the uterine cavity are now displayed.

MEDITERRANEAN (COOLEY'S) ANÆMIA IN AUSTRALIA.

By A. H. TEBBUTT,
Sydney.

RECENTLY in this journal (1949) Sinn reported a case of Cooley's anæmia and said that no other cases had been reported in the Australian medical Press. His case was that of a boy, first examined at the age of seven and a half years, and at the time of his report eleven and a half years old. Though the boy was born in Australia, both of his parents came from an island near Greece.

This communication deals with two adults, both of Mediterranean stock, who presented a chronic target-oval cell anæmia, microcytic and hypochromic, without any ascertainable ætiology and refractory to hæmatinics. Included is the blood picture of the father of one of these patients. Several American workers, for example Dameshek (1943) and Smith (1943), have shown that families of Mediterranean origin, notably Italians and Greeks, may present syndromes varying in severity from Cooley's erythroblastic anæmia to a mild hypochromic anæmia in which target and oval cells and increased resistance to hypotonic saline are prominent features. The red cell trait is familial and apparently inherited as a Mendelian dominant or a partial dominant. In some cases of Cooley's anæmia, both parents have shown the milder syndromes associated with the trait, and the suggestion is that the severest syndromes met with in childhood and usually fatal before the age of twelve years are accounted for by

homozygosity as regards this trait, whereas persons with the milder forms are heterozygous.

It should be borne in mind that target cells are not peculiar to Mediterranean anæmia, for in fact they were first described and given this descriptive title by Barrett (1938), who found them in the blood after splenectomy and in such diverse conditions as obstructive jaundice, steatorrhœa and hypochromic anæmias. But later in the same year target cells were identified in Mediterranean anæmia, and they have since become identified with it more specifically in the literature than with other conditions. It is apparently the shape and thinness of the cells (leptocytes) which are characteristic, and according to Barrett a bowl shape instead of biconcavity gives rise to the target picture in dried and stained films. Oval cells are often conspicuous. Anisocytosis, anisochromia and poikilocytosis are usually prominent features, there is an increase in reticulocytes and basophilic stippling is often described. Though microcytosis predominates, macrocytes, usually hypochromic, may be present. Erythroblasts are often numerous in the child patients and may be present or absent in affected adults.

The anæmia is of the hypochromic microcytic type, but there may be polycythæmia with lowered colour index; this has been explained as an attempt to compensate by numbers for the often greatly reduced mean corpuscular hæmoglobin figure. The leptocytosis is associated with increased resistance to hypotonic saline, and this is in accord with Haden's theory that the thicker the red cell, the greater its fragility. In contrast to the spherocytes of congenital hæmolytic anæmia (familial acholuric jaundice), the target cells have a large surface area relative to volume and are said to be capable of taking up much fluid before rupture.

Smith (1943) found that the mildest forms were without symptoms, and that the subjects appeared to be in good health and would escape detection without a careful blood examination. The skeletal changes common in Cooley's anæmia are usually absent or dubious in affected adults. Though leucocytosis is usual in Cooley's anæmia and may be high—from 13,000 to 50,000 leucocytes per cubic millimetre according to Whitby and Britton—and although some immature myeloid cells may be present, yet the milder syndromes both in children and in adults seem to present either normal leucocyte numbers or only mild to moderate increases.

Case I.

The first case is that of a married woman, aged thirty to forty years, who has suffered from anæmia, varying in degrees but with a fairly constant low colour index (from 0·6 to 0·7), for at least four years. It has not responded specifically to hæmatinics. Her father and mother were both born in Lebanon and belong to the Lebanese race. A recent blood examination shows some of the essential features of the dyscrasia. The red blood cells numbered 5,300,000 per cubic millimetre, the hæmoglobin value was 9·9 grammes *per centum*, and the colour index was 0·6; the hæmatocrit reading was 34%. The calculated mean corpuscular volume was 63 cubic μ, the mean corpuscular hæmoglobin content was 18 micromicrogrammes and the mean corpuscular hæmoglobin concentration was 29%. The leucocytes numbered 7,700 per cubic millimetre, 7% being band forms and 50% segmented forms (total neutrophile cells 57%), 33% lymphocytes, 8% monocytes, 1% eosinophile cells and 1% basophile cells. These figures showed a considerable disproportion between the number of red cells and both the hæmoglobin value and the hæmatocrit reading, the greater reduction being in the hæmoglobin value, so that the anæmia is both microcytic and hypochromic. The stained films showed numerous microcytes, poikilocytes, target cells, oval cells and macrocytes with central pallor. Reticulocytes were increased in number and many had a dense reticulum. A very few normoblasts were found. The blood serum gave a prompt reaction in the indirect Van den Bergh test. The red cells showed increased resistance to hypotonicity, hæmolysis commencing in 0·4% sodium chloride solution and being incomplete in 0·27% solution, whilst two specimens of control blood gave complete hæmolysis in 0·34% sodium chloride solution with the same set of dilutions.

The father of this patient was apparently in good health, but examination of his blood showed the microcytic and hypochromic and target-oval cell trait. He was a man of about sixty years, and the blood picture was as follows. The red blood cells numbered 6,400,000 per cubic millimetre, the hæmoglobin value was 12·9 grammes *per centum*, the colour index was 0·67 and the hæmatocrit reading was 41%; the calculated mean corpuscular volume was 67 cubic μ, the mean corpuscular hæmoglobin content was 20 micromicrogrammes, and the mean corpuscular hæmoglobin concentration was 31%. The leucocytes numbered 7300 per cubic millimetre, 7% being band forms and 47% segmented forms (total neutrophile cells 54%), 33% lymphocytes, 7% monocytes, 4% eosinophile cells and 2% basophile cells. These figures show an increased number of red cells but a reduced hæmoglobin figure, so that the colour index is low, and the mean red cell volume and mean corpuscular hæmoglobin content are also reduced and the mean corpuscular hæmoglobin concentration is slightly reduced. It is a microcytic and hypochromic anæmia. In stained films the red cells showed slight anisocytosis, but there were oval cells, a few poikilocytes and a very few target cells. No nucleated red cells were found. The tendency to hypochromic polycythæmia is seen in this case. A red cell fragility test was unfortunately omitted.

The above findings leave little doubt that this is a familial anæmia, with clinical expression in the daughter, but asymptomatic in the father. If the mother also showed the target-oval cell trait, then it is possible that the daughter is homozygous, though not necessarily so.

Case II.

The next patient presents some puzzling features, and the diagnosis of uncomplicated Mediterranean anæmia is somewhat hazardous; but I can see no more satisfying explanation for the background of his blood dyscrasia. He is a man, aged fifty-four years, of the Greek race and born in Greece, has had splenomegaly and hepatomegaly and a blood dyscrasia for at least three years. A recent blood examination shows some of the features of this dyscrasia. The red blood cells numbered 3,900,000 per cubic millimetre, the hæmoglobin value was 8·6 grammes *per centum*, the colour index was 0·74 and the hæmatocrit reading was 27%; the mean corpuscular volume was 69 cubic μ, the mean corpuscular hæmoglobin content was 22 micromicrogrammes and the mean corpuscular hæmoglobin concentration was 32%. The leucocytes numbered 28,000 per cubic millimetre; 6% were band forms and 76% segmented forms (total neutrophile cells, 82%), 8% were lymphocytes, 2% monocytes, 8% eosinophile cells, and no basophile cells were present. In stained films anisocytosis and anisochromia were present. There were many oval cells, target cells, poikilocytes and polychromasic cells. There were also large cells with pale centres and large normochromic cells, also fully hæmoglobinized microcytes suggesting spherocytes. One normoblast was found. Reticulocytes were increased in number, and those with a heavy reticulum were prominent. Platelets were very numerous and formed conspicuous clumps. The fragility test revealed an increased resistance to hypotonic saline, hæmolysis commencing at 0·4% sodium chloride solution, and being not quite complete at 0·24%. With the salt solutions used a control specimen of blood gave commencing hæmolysis at 0·45% and complete hæmolysis at 0·37%. The neutrophile leucocytosis was peculiar and unlike that of infective conditions, for there was no "shift to the left" and no significant degenerative or "toxic" changes. Cells with five and six lobes were not uncommon (hypersegmentation). Leucocytosis is not uncommon in Cooley's anæmia in children, but the figure in this case is uncommonly high for the form of the dyscrasia met with usually in adults. It does rather suggest an obscure myeloid metaplasia of spleen and liver without a leuchæmic blood picture; but the red cell morphology, the low colour index and the increased resistance to hypotonicity are suggestive of the Mediterranean dyscrasia in a person of the Greek race.

I am indebted to Dr. G. A. W. Johnston for allowing me to see a sternal biopsy section prepared from this patient at Sydney Hospital. It shows cellular marrow with increase of megakaryocytes, and amongst the more immature myeloid series are numerous leucocytes.

It may be that the high leucocytosis and the thrombocytosis in the peripheral blood are only unusual and atypical features in a severe and chronic case of Mediterranean anæmia, though I admit the difficulties in this diagnosis. A second interpretation is that this is another type of bone marrow dyscrasia with splenomegaly and hepatomegaly and megakaryocytosis, possibly a myeloid metaplasia, arising coincidently in one of the Greek race with the background of the familial target-oval cell syndrome.

Comment.

These cases are reported because there has been much Mediterranean immigration into Australia, and the syndrome will certainly be met with if borne in mind or looked for in the races most affected, as it has been in the United States—to which country we owe a debt in this connexion. As Wolman and Dickstein (1946) point out, the mild or minor form is beginning to be recognized as widely prevalent in contrast to the marked or major form, which has been longer and better recognized, but is more uncommon.

Summary.

Several cases are reported of anæmia in adults which appears to be Mediterranean (Cooley's) anæmia.

Acknowledgement.

I am indebted to Dr. A. J. Hood Stobo for permission to report on a patient under his care.

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Reviews.

ELEMENTARY ANÆSTHESIA.

ONE could hardly imagine a more concise and informative work than Gordon Ostlere's small book, "Anæsthetics for Medical Students".[1] Within the compass of but 105 pages, and employing the utmost economy in words and detail, he describes most effectually the several basic and practical factors involved in modern anæsthesia. If intended only for undergraduates, the book perhaps exceeds their limited understanding and capabilities; in some respects it implies a degree of initiative and responsibility quite beyond their capacity. However, it is felt that the term "students" means both the unqualified and the qualified in medicine, so that the information provided should be invaluable for hospital resident medical officers.

The style is simple, direct and enlivening, thus inviting sustained interest and abolishing tedium in a perusal of the work. Although brevity has introduced a few obscurities and inadequacies, the general outline is admirable. If false emphasis, both positive and negative, occurs from time to time—for example, references to the "motionless flaccid field" and "extremely light anæsthesia"—most points are very well elucidated. A notable feature is that the book fits easily into the coat pocket, making for its ready and frequent accessibility.

The influence of current English practice is evident in the work, in that pride of place is given to the use of nitrous oxide, oxygen and ether by the "semi-closed" method. In our country, of course, "open" ether would enjoy such priority both for teaching purposes and for common applicability. Further, the liberal dosages of atropine recommended (grain 1/75) would seem to favour the onset of convulsions or atelectasis in some cases. However, such admonitions as "if you can't be good, be careful" and "if in doubt use ether" are highly valid anywhere.

Opening with a discussion of general methods and requirements, the book proceeds to a simple assessment of the patient, under the striking headings of the fit, the young, the old and the ill. Next comes an excellent study of premedication, followed by a thorough account of the complications likely during the induction of anæsthesia. The signs of anæsthesia are then dealt with satisfactorily enough for learners, despite some confusion about the eyeballs and pupils. While the latter may exhibit intrinsic movements they certainly do not "squint"; this is the embarrassing prerogative of the former. The various common anæsthetic agents now receive attention; there is an excellent section on the "intravenous" barbiturates, which closes with brief mentions of cyclopropane and curare. Apparatus, endotracheal anæsthesia, the prevention and treatment of shock, and the chief post-operative complications are next considered. These are followed by some good notes on local and spinal analgesia. The final two chapters largely review the preceding matter, while a satisfactory index completes the work.

A few errors need comment. Chloroform is not the only agent likely to cause primary cardiac failure. "Avertin" is not made up as a 2·5% solution in amylene hydrate. Misguided premedication does not necessarily "ruin" the subsequent anæsthetic. When complete abdominal relaxation is produced by curare the diaphragm cannot maintain adequate respiration. The fall of blood pressure in high spinal analgesia is not due to splanchnic paralysis. To pack the pharynx during anæsthesia, even endotracheal, for laparotomy in intestinal obstruction is a grave error. Finally, "ophthalmic" is not spelt "opthalmic", a mistake quite excusable, since it is the only one noted.

[1] "Anæsthetics for Medical Students", by Gordon Ostlere, M.A., M.B., B.Chir. (Camb.), D.A., with a foreword by C. Langton Hewer, M.B., B.S., M.R.C.P., F.F.A.R.C.S.; 1949. London: J. and A. Churchill, Limited. 7¼" × 4¾", pp. 116. Price: 7s. 6d.

TROPICAL DISEASES.

WE are told in the preface to "Diseases of the Warm Climates"[1] that this edition in English, made in the United States of America for the London publishers, was produced simultaneously with the Belgian edition, which is written in French. It would seem that Professor Dubois and Professor Van den Berghe were badly advised in this respect. The English text is so poorly presented, and yet contains so much valuable material, that one longs for a look at the French text; it is quite obvious that the English one does not do it justice. In this edition, the historical notes are crisp summaries in excellent English; most of the remainder is in good English, marred by the somewhat confusing inclusion of definite articles in the French style; but every here and there are sections full of grotesque word-for-word translations of French idioms and rashly anglicized French words. In these last sections it is often difficult to decide what is meant; in them, also, appear most of the numerous misprints. It is unusual, too, to see so many American modifications of spelling over a London imprint; even these are not constant—"amebic" and "amœbic" appear in the same paragraph.

All of this is most disappointing. The authors show themselves to have wide knowledge and ample experience. Apart from their tendency to generalize occasionally when more practical details would be welcome, they have a valuable contribution to make, and give the impression that the French edition would be well worth studying. It is impossible to recommend the English edition because of its many imperfections.

[1] "Diseases of the Warm Climates: Their Clinical Features, Diagnosis and Treatment", by Albert Dubois, M.D., and Louis Van den Berghe, M.D., D.Sc.; 1948. London: William Heinemann (Medical Books), Limited. 9" × 5¾", pp. 468, with 100 illustrations. Price: 42s.

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ANIMAL MECHANICS.

IN 1873 there was published a book entitled "Principles of Animal Mechanics", by the Reverend Samuel Haughton, F.R.S. The author was a trained mathematical physicist, and it may be truly said that nothing like this work had appeared before, though the methods and principles of physics had of course been applied to the physiology of the ear and eye and by a few anatomists to the movements of the joints. Haughton was not content with existing anatomical material, human and comparative; he dissected a large number of vertebrate animals and was here fortunate in having at his disposal the famous Zoological Gardens of Dublin. The numerous forms in which muscular fibres can be arranged were examined by him in detail and were classified under nine headings, each being subjected to mathematical analysis, which, when applied to skew and ellipsoidal surfaces, demanded a discipline of training in spherical geometry which falls to the lot of only a few. The book was before its time and made little impression on medical readers whose physics and mathematics were merely that sketchy introduction which was then given in the first year of medical study. Haughton not only analysed the direction and intensity of the forces exerted during muscular action, but calculated in certain instances the energy and that other dimension, namely power, which at a later date investigators of calorie requirements almost invariably forgot and which even today figures much too little in estimates of mechanical work derived from food energy. Haughton was also the first to apply the physical "principle of least action" to contractile processes, notably the systole of the heart, and he also drew attention to the elastic and extensile properties of the coats of arteries which resemble, as he put it, the concentric layers in modern artillery. It is rather singular that in a work displaying such acumen a few errors should have slipped in, particularly the attempt to measure the blood flow by the spurt of blood when an artery was cut. Here he made the mistake, very common amongst laymen, of regarding the flow of blood in an artery as rapid; as we now know, the initial trajectory of blood from a cut vessel arises from the release of fluid under pressure in a distended and elastic container and would be equally great if the blood were stagnant. But these are merely spots in the sun. Subsequent research on muscular contraction was concerned chiefly with the supply of chemical potential energy and the nature of its transformation into work—a problem by no means yet solved. In 1912 the physicist W. H. White could state that the specification of every prime mover should include starting effort, best speed, greatest power at that speed and greatest power obtainable and for how long without injury. Muscles, he went on to say, are adaptable in a manner unknown to engineering, for there is no such thing as a gearbox. "A man can exert ¼ horsepower running up stairs, his usual being ⅛ horsepower." Further research by A. V. Hill has demonstrated that there is actually a best speed, though this is not easy to determine, and further, that the horsepower exerted by an athlete can, for a brief period, be actually greater than unity. This adds all the more to our admiration of muscular tissue as a prime mover.

The contributions of physics to biology have in recent years taken quite another direction, being mainly occupied with the unravelling of the problems of cellular morphology and metabolism. The microscope had remained virtually unchanged from 1880 to 1940, but soon there came ultra-violet, infra-red, polarizing and reflecting microscopes, and these had to yield in resolving power to the electron microscope which could make visible units containing a hundred or less atoms. Infra-red spectroscopy combined with X-ray crystallography now gives a picture of atomic pattern undreamed of before. Here it may be said that the heavy and slow labour of computation has been simplified by the use of electronic calculating machines. The ultra-centrifuge of Svedberg, electrophoresis of Tiselius and light scattering of Doty have yielded a quantitative representation of the large molecules which play so important a part in biochemical happenings. Radioactive tracer isotopes have demonstrated the rate of flux of material into and out of living structures and made more felicitous than ever T. H. Huxley's simile of a wave in a rapid. But perhaps most important of all is the development of quantum physics. That light energy is radiated in discrete units or quanta was at first an abstraction of the higher physics and even today is applied with difficulty to the radiant energy employed in wireless; but its effect was revolutionary in physics and is now becoming revolutionary in biology.

In the address by Professor J. D. Bernal on the physical basis of life,[1] an utterance of outstanding significance in modern biology, there occurs the following: "Without exaggeration it can be said that the conception of quantum energy changes in chemical reactions is the most illuminating and the most effective new idea in modern biology." And again: "The key to the understanding of this chemical evolution of life lies in the junction between observational biochemistry on the one hand and quantum theory on the other." It is interesting to note in this connexion that quantum considerations have been applied recently to photosynthesis. No more than four quanta are required to produce one molecule of oxygen gas; some measurements suggest only three quanta. The figure "4" means an

[1] J. D. Bernal (1949), "The Physical Basis of Life", *The Proceedings of the Physical Society*, Section A, Volume LXII, Part 9, September 1, page 537.

efficiency of 65%, whilst "3" would mean 85%.[1] Whilst we are thankful for this magnificent extension of analysis and measurement, we can still suggest that there is room for further researches in the direction of Haughton's animal mechanics. Take for example shock absorption. There is an authenticated story of a small Australian boy being taken to a zoo and gazing with admiration at an elephant walking. "Isn't it well sprung?" he remarked. The youngster was right; the elephant is exceedingly well sprung. Apart from special mechanisms in the foot, the quadrupedal mammal in most cases uses the constantly flexed hock and stifle as efficient shock absorbers; in the forelimb the device of receiving the impact on the floating shoulder blade, which is braced by the powerful pectoral muscles, is admirably adapted to locomotion and jumping. In the human being most of this is lost; the arch of the instep and the thin articular cartilages of ankle, knee and hip absorb but little of the jolt sent on to the sacrum; above this the elastic intervertebral disks save the skull from basal fracture, but not always. It is significant that the skier adopts a posture in which flexed knees and hips function like the hock and stifle of the horse and allow jumps to be undertaken which would be dangerous or even fatal in the ordinary erect posture of walking. The application of engineering considerations to bone has recently been undertaken, especially with reference to the stresses received in parachute falls. The director of the American National Bureau of Standards announced in the *Journal of the Franklin Institute* that long bones have twice the tensile strength of hickory.[2] He did not mention that precisely the same figure had been obtained fully twenty years ago. Anyhow, bone with its interlocking systems composed of concentric hollow cylinders tied together with radial rivets and reinforced with mineral matter in which calcium phosphate preponderates, may be described as an engineering marvel. Rapid travel by jet-propelled plane or rocket has already directed attention to several problems of applied physics and physiology which need investigation; amongst these mention should be made of the Coriolis force which Professor R. W. Pohl, of Göttingen, as far back as 1932, was demonstrating in class experiments, but which even today suffers from a curious neglect in English-speaking countries. Pohl in 1937 was investigating the Coriolis force in human beings in rapid flight, but the results of his researches have not been disclosed, probably for military reasons. When Bleriot in his primitive plane crossed the English Channel in 1909 he made the very prophetic remark that the only limits imposed on air flight would be those imposed by the human body. One of the tasks of animal mechanics is to make a correct assessment of these limits.

[1] Dean Burk *et alii* (1949), "The Maximum Efficiency of Photosynthesis: A Rediscovery", *Science*, September 2, page 225.

[2] "Notes from the National Bureau of Standards, Mechanical Properties of Human Bones" (1949), *Journal of the Franklin Institute*, June, page 597.

Current Comment.

THE ANÆMIAS OF MEDITERRANEAN COUNTRIES.

SOME of the transmissible blood disorders are sufficiently rare as to warrant their omission from the smaller text-books of medicine. Some of them, also, affecting special races, such as sickle cell anæmia, or populations of limited areas, like Cooley's anæmia, seem remote from a country like Australia. Now, however, with the contraction of the time-space relation of the world due to the speed of modern transport, and the upsetting of ethnological boundaries by the disruption of war, we cannot affect aloofness to such conditions. In any case, hæmatology is such an important branch of general medicine that knowledge gained from any source may at any time have a practical bearing on such diverse problems as inherited characteristics of the blood, and toxic effects of chemicals used in industry or in therapeutics. A critical survey of one important group of hereditary blood diseases has been published by V. Chini and C. M. Valeri, which draws upon no fewer than 277 references to literature, chiefly of European origin.[1] These authors think that after ten years of investigation they can classify the anæmias of the type described by Cooley, and allied varieties. In addition to Cooley's type, which affects children, they mention another syndrome widely recognized in Italy during the past generation, and group these together under the heading of "Mediterranean hæmopathic syndromes". The interest of the latter of these conditions lies in its association with hæmolytic jaundice with decreased red cell fragility. The red cells usually have an oval shape, and are also often small and irregular. This syndrome has naturally been of special interest to Italian investigators, and Chini and Valeri consider that it is really a mild form of Cooley's anæmia, which permits the subjects to reach adult age. This concept allows a simple classification: a severe type of anæmia affecting children, usually rapidly fatal, and a mild one not uncommon in adults. In the former or classical type the anæmia is intense, the spleen is enlarged, with metaplasia affecting the red cell formative mechanism by changes in the bones. These bony lesions are distinctive and have been of great palæontological importance, since they can be recognized in skeletons or mummies of ancient races, including the Aztecs, the Incas, Indians from Colombia and Yucatan, Egyptians, and palæolithic remains found in caves and elsewhere in Italy. Diffuse osteoporosis and radial striation of the skull have been demonstrated in these skeletons. These findings have been made all the more significant by the finding of sickle cell anæmia among some native populations in Mexico whose anthropological characters resemble strongly those of the ancient Aztecs. The suggestion has been made that these changes have not only been associated with blood disorders, but that therein may lie a reason for the physical and social decay of these vanished peoples.

Returning to the present day, Chini and Valeri have first established common and distinctive features in their case material, and then assuming a single generic clinical picture, have essayed to explain the pathogenesis. Briefly, they point out that among the families of sufferers from Cooley's anæmia blood changes are frequently found, the most common being an increased resistance to hypotonic solutions. The same is often seen in siblings as well as parents. Carriers of this syndrome are found in those regions where Cooley's anæmia is endemic. But in many instances of this "Mediterranean hæmopathic syndrome" decreased fragility of the red cells appears; this is, of course, readily recognized clinically because of the occurrence of hæmolysis, with jaundice, splenomegaly and intense blood crises. Removal of the spleen relieves the hæmolysis, but other changes are unaffected, and the picture then resembles more the classic Cooley picture, even with osteoporosis. For these reasons the apparently different conditions are believed by the authors to be essentially the same, the milder one in adults being necessary for the appearance of the severe, fatal type in children. One important point has been established, that both parents of such a child always show blood changes: there is thus reason for regarding Cooley's anæmia as an example of a dominant hereditary characteristic with a lethal homozygous effect. When one parent only has been affected the result is the milder and not the severe form of the disease. Inquiry has been made into some of the obscure features, but as yet it is not known why the

[1] *Blood*, September, 1949.

carriers should acquire a hyperhæmolytic state, or what is the significance of other morphological changes, like ovalocytosis. These matters must be left to the experts, but in the meantime here is a disease which is of great social importance, for its incidence is high in certain areas, and there is evidence that it has existed for many centuries. Caminopetros has given a warning that this hereditary disease will inevitably spread, owing to its dominant characters, and possibly this warning may be recalled later in other parts of the world relatively remote from that sea which has been in the centre of the stage for so long—the Mediterranean.

MEDICAL ASPECTS OF HIP FRACTURES IN OLD PEOPLE.

THE medical problems associated with the management of hip fractures in elderly patients are only too well known to orthopædic surgeons, physicians and general practitioners. The remarkable results that have followed the bold adoption of nailing are also well known, and many old people with pronounced medical disabilities have survived the operation under modern conditions and have been spared the dangers of prolonged immobilization. However, as is pointed out by R. J. Fahey, R. M. Kilfoyle and J. H. Shortell,[1] the most important medical factors have not been emphasized in the literature on hip fracture; criteria have not been established regarding selection of the patient and the time of operation or definite measures determined to be taken under specific circumstances to facilitate surgery or to make it possible. Thus deaths have occurred post-operatively among patients who were judged acceptable for hip nailing—usually from conditions that were present before the operation or even before the fracture occurred. To clarify their thoughts in the matter, Fahey and his colleagues have studied the records of 100 consecutive patients, aged seventy years or over, who were treated on the Sixth Surgical Service at Boston City Hospital for fracture in the region of the hip; 61 patients had intertrochanteric fractures, and 39 fractures of the femoral neck. Seventy-two patients were operated upon. The remaining patients were treated by traction or the application of a plaster spica. Conservative treatment was instituted because of the personal choice of the surgeon, because of the nature of the fracture, or because the patient was considered a "poor surgical risk". Among the patients operated upon the mortality rate was 18%. On the basis of the history, physical examination, laboratory studies and response to therapy these patients have been classified into three groups, depending upon the amount and type of concomitant medical disability observed during the first few days in hospital. In the first group, which comprised 13 patients, were placed those who, in spite of their age, were in good physical condition. There was no mortality in this group, which is as one would hope it should be. In the second group were placed patients who had underlying physical disabilities that required active medical treatment before and after operation. The fact that hip fracture in the elderly is very often as much a medical as a surgical problem is well exemplified here, as seventy patients came into this group. Four patients in this group died. The third group comprised patients with severe and extensive medical disabilities who, under adequate medical management, had shown no evidence of improvement or were becoming worse under conservative treatment. Seventeen patients fell into this group; nine were operated upon and eight were treated conservatively; all died. These facts certainly appear to support the belief of Fahey *et alii* that if a patient is becoming worse under adequate medical management in traction, operation will not in any way alter the course for the better; on the contrary, it will hasten death. They consider that patients in this third group, but only they, should be refused operation.

Forty-one of the patients in this study had either arteriosclerotic or hypertensive heart disease or both; 10 of these 41 patients were suffering from congestive cardiac failure. The important point appears to be that the presence of cardiac disease has little or no bearing on the general mortality rate, provided there is no failure. Failure, if present, must be corrected before operation is attempted, but once this has been done no type of heart disease, except recent myocardial infarction, is a contraindication to hip nailing. Another point to be remembered is that in the aged the manifestations of peripheral venous thrombosis and pulmonary embolism may be minimal and may be unrecognized unless sought for diligently. Less common than cardio-vascular disorders, but still important, are pulmonary complications. All care is needed to prevent and to control these; the use of penicillin before and after operation has been a tremendous help. Renal factors also deserve special attention amongst the elderly. If the non-protein nitrogen content of the blood is high or the carbon dioxide combining power of the blood is low, correction of the condition is necessary before operation by the use of fluids, sodium lactate or bicarbonate and saline solutions as indicated. If there is no chemical improvement operation should not be performed. There is a good deal of discussion of the management of medical conditions in this paper, which is worthy of more detailed perusal by those interested. It is important that a worthwhile surgical advance should not be spoilt in any way by deficiencies of medical management.

[1] *The New England Journal of Medicine*, September 1, 1949.

THE HEALTH OF NURSES IN HOSPITAL.

APART from the question of tuberculosis, surprisingly little attention has been paid to the health of nurses, at least in any systematic or comprehensive way. Even in Britain, according to Donald Court,[1] who has been looking into the matter, an exact measure of the risk to health in nursing has still to be made. He states that surveys by the American Hospital Association suggest that it is considerably greater than among college students and teachers—"a reasonable expectation when adolescents and young adults are continuously exposed to disease"—and his own impressions are that the health of hospital nurses, especially student, assistant and auxiliary nurses, is less satisfactory than the health of women in other professions. This is based on a survey in an urban hospital and comparison of the results with those of similar limited investigations. There is no call to discuss the survey in detail here, though its careful study would be of value to those interested; but it was, in brief, a study of the health of 300 nurses during the three years from 1943 to 1945. The main causes of excessive ill health were respiratory infection, skin sepsis, infective diarrhœa, infectious fevers and tuberculosis. Of the 300 nurses, 18 (6%) developed tuberculosis during the survey, with the loss of 3000 working days; there were no deaths from this disease during the three years. Court outlines measures which should help in this problem. He discusses commonsense principles and practical methods in hospital practice designed to reduce the exposure of nurses to tuberculous and other infections; a plan for the supervision of nurses' health by initial and subsequent examinations and immunization methods; the provision of satisfactory facilities for treatment of nurses during illness, and sensible ideas on health education. Again the details need not concern us; some are of local interest, many would apply generally, but before we can adapt and adopt the ideas put forward we need basic data from first-hand surveys of the question in our own country. One notable finding from Court's survey may spur us on. Shortage of nurses is still a serious practical problem and a handicap to the provision of the best medical treatment. Court found that ill health played a large and unsuspected part in wastage; 17% of those leaving the hospital and 25% of those leaving the profession (at least temporarily) did so because of ill health. Apart from this, illness means loss of time, which is costly to hospital, nurse and patient. A practical approach to the problem, based on reliable local data, would pay all-round dividends.

[1] *The Lancet*, November 12, 1949.

Abstracts from Medical Literature.

OPHTHALMOLOGY.

Treatment of Glaucoma.

LOUIS LEHRFELD (*Archives of Ophthalmology*, September, 1948) maintains that every patient with glaucoma should be treated medically until such time as control of intraocular pressure is no longer maintained at a limit at which visual acuity and visual fields may be conserved. Acute congestive glaucoma should be treated intensively medically for twenty-four hours and surgery resorted to if tension has not been reduced. However, if reduction in tension under intensive miotic therapy is such as to bring about a return to normal vision, surgery may be deferred. The treatment of chronic congestive glaucoma is medical. Control of intraocular tension in this category does not mean a reduction to normal, but consists in reduction to a degree which the eye in question will tolerate. If the patient has repeated exacerbations of increased intraocular pressure with symptoms, operation should be performed. The author recommends a basal iridectomy if the eye is red, an iridencleisis if it is only mildly injected, and an Elliott trephination if it is "quiet". For chronic simple glaucoma he prefers medical treatment even if the vision is not normal, the visual fields are cut down and the tension is not normal. He finds that 70% of all patients with chronic simple glaucoma can be made comfortable, and surgery is performed when miotics are failing and the patient is losing his vision; in these cases the operation of choice is iridencleisis. He makes the point that before operating on patients with chronic simple glaucoma the surgeon should remember that old people prefer a little sight with glaucoma to a blind eye that apparently has been cured of glaucoma.

An Operation for Ptosis.

RAYNOLD N. BERKE (*Transactions of the American Academy of Ophthalmology and Otolaryngology*, May, 1949) describes a new operation for ptosis, recommended for cases in which resection of the levator muscle is not indicated. Specifically it is useful in four types of ptosis: (i) complete ptosis with normal superior rectus muscles; (ii) complete ptosis with weak superior rectus muscles; (iii) ptosis associated with the jaw-winking phenomenon; (iv) complete ptosis associated with blepharophimosis. It consists of an operation on the globe to shorten the superior rectus muscle combined with an operation on the lid to correct the ptosis. In spite of the fact that the superior rectus muscle is shortened, the author has operated on four patients with a normal superior rectus muscle without the production of overaction or diplopia. In ten cases in which the superior rectus muscles were weak there was lessening of the ptosis and the hypotropia was decreased. Good results were also obtained in five cases of the jaw-winking syndrome. In this operation the whole width of the superior rectus muscle is transplanted onto the anterior surface of the tarsus. The advantages of the operation are that the superior rectus muscle can be used even though paralysed, slipping of the transplanted tendon is reduced to a minimum, a smooth curve of the upper lid results, turning-in of the upper lids is prevented, a good upper-lid fold is produced, post-operative hypotropia is prevented, the operation can be used in the very young, and the lid moves synchronously with the globe. The only disadvantages claimed are that normal winking is interfered with and lagophthalmos is present during sleep.

Cauterization of the Ciliary Body for Glaucoma.

S. J. MEYER (*Archives of Ophthalmology*, April, 1949) considers that cyclodiathermy puncture is indicated in cases of flat anterior chamber, especially after trephination or iridectomy, aphakia with secondary glaucoma, secondary glaucoma due to prolapse of the iris, primary glaucoma in which the angle of the anterior chamber is obliterated, and finally hæmorrhagic glaucoma, and that the result may be considered successful if the patient is comfortable and relieved of pain. The results are inconstant in complicated or secondary glaucoma. The post-operative hypotension may be only temporary, the tension returning to its previous pre-operative level, the post-operative hypotension may progress to atrophy of the eyeball, or, in certain cases, normal intraocular pressure is attained and maintained. After operation visual acuity returns to its previous state, and in cases in which examinations of the visual field were possible, no decrease in the retinal function was noted. The dangers and complications involved in this operation concern mostly the cornea and lens.

Sarcoidosis.

A. C. WOODS (*Transactions of the American Academy of Ophthalmology and Otolaryngology*, March, 1949) briefly describes the ocular manifestations of sarcoidosis. He states that the disease is of particular interest to ophthalmologists because 50% of all patients with sarcoidosis have ocular involvement. Uveo-parotid fever and Mikulicz's syndrome are ocular manifestations of sarcoidosis as are 5% to 10% of cases of uveitis. The skin of the lids may be involved, small nodules like millet seeds appearing. In the lachrymal gland sarcoidosis is manifested as a painless swelling of the outer and upper portion of the orbit. Usually lachrymal gland involvement is associated with enlargement of the salivary glands and cervical lymph glands. Only very rarely is the conjunctiva involved, and the cornea and sclera are involved not at all. Nodules may appear in the episclera over the insertion of the rectus muscles. Iritis is the most frequent and important ocular manifestation, the most characteristic picture being a nodular iritis, usually painless and with only slight inflammatory symptoms. In some cases there is associated parotid swelling. Chorioidal and retinal forms of sarcoidosis are rare.

B. L. GIFFORD AND A. C. KRAUSE (*Archives of Ophthalmology*, June, 1949) give a full account of sarcoidosis with special reference to ocular signs. They state that as the ophthalmologist may be the first to examine patients with this condition, an appreciation of the disease is necessary. The patient with sarcoidosis is seldom acutely ill, and it may be only ocular symptoms which bring the patient to seek advice. An examination of the eyes may reveal any combination of nodules of the lids, conjunctivitis, keratitis, iritis, iridocyclitis and rarely chorio-retinitis. The severest and most obvious feature is generally iritis of a seroplastic type, with posterior synechiæ and abundant keratic precipitates of the mutton-fat type. In the presence of skin lesions and possibly enlarged lymph nodes, sarcoidosis should be suspected and considered. Lesions in the chest are not necessarily characteristic, but they are probably the most frequent clinical sign and are of two general types, either involvement of the hilar nodule consisting of gross enlargement of the bronchial nodes, with coarse radiations of dilated, deep lymphatic striæ extending from the enlarged nodes towards the periphery of the lungs, or involvement of the hilar nodules accompanied or followed by widespread miliary lesions of irregular size throughout the lungs. The two types may be present together. Lesions of bone are characteristic but rare. They are seen in the metaphyses of the phalanges and in the metacarpal bones of the hands and feet. They are definite punched-out lesions with sharp borders. The cutaneous lesions fall into three types: disseminated miliary sarcoidosis, subcutaneous sarcoidosis and *lupus pernio*. A negative result from the Mantoux test is of value in the diagnosis of sarcoidosis, and the authors regard the presence of a positive result with a dilution of 1:1000 old tuberculin as throwing doubt on the diagnosis of sarcoidosis. Therapy is directed toward empiric treatment of the iritis. General treatment consists in a modification of the regimen ordered for tuberculosis. Calciferol in doses of 50,000 units three times per day holds some promise. In one case an extremely painful iritis became painless and definite clearing of the aqueous flare began shortly after treatment with calciferol was commenced. The authors consider that the most important requirements and investigations for a diagnosis of sarcoidosis are a clear history of the disease, clinical evidence of diseases simulating sarcoidosis, low cutaneous sensitivity to old tuberculin, biopsy of the skin, lesions of bone, X-ray examination of the chest, biopsy of lymph nodes, the gross appearance of cutaneous lesions, examination of the blood for albumin-globulin ratio, and ocular examination, in that order of reliability.

Ocular Complications of Temporal Arteritis.

CARROLL SMITH AND PHILLIP GREENE (*American Journal of Ophthalmology*, May, 1949) draw attention to ocular improvement in temporal arteritis and report four cases. They state that this disease should be borne in mind when an elderly patient suffers an occlusion of the central retinal artery or its branches, particularly if there is a history of head and scalp pains, painful nodules along the course of the temporal arteries and absence of pulsation. They point out that there is an unusual discrepancy between the visual loss and objective findings. In some cases there is obstruction of the central retinal artery, but the impression may be given of an interruption of the vascular nutrient branches to the optic nerve finally producing the picture of cavernous atrophy and excavation of

the nerve. Probably the interruption of the blood flow comes on gradually, so that the typical picture of central artery embolus is not seen. The disease is probably a widespread one and the ophthalmic artery is involved directly or from central vessels.

Diathermy for Recurring Retinal Hæmorrhage and Retinitis Proliferans.

F. H. VERHOEFF (*Archives of Ophthalmology*, September, 1948) describes the successful use of diathermy for recurring retinal hæmorrhage. Surface diathermy was applied over the affected region of the retina; ophthalmoscopic examination then showed extensive white areas in the region treated. The treatment was repeated nine months later. Fifteen months after the last application of diathermy there had been no new hæmorrhages; corrected vision was normal, but there was contraction of the visual field of the treated eye. The author suggests that diathermy may be beneficial in cases of retinal vascular obstruction from any cause. It may establish communication between retinal and chorioidal vessels and thus restore retinal circulation.

Melanoma of the Optic Disk.

F. C. CORDES AND M. J. HOGAN (*American Journal of Ophthalmology*, August, 1949) feel justified, because of its rarity, in reporting a case of primary malignant melanoma of the optic disk. Five proven cases have previously been reported. The tumour was found by accident in a Negress, aged fifty-two years. Microscopic examination showed that the growth appeared to arise from the tissues of the optic nerve anterior to the *lamina cribrosa*. The possible sources of origin of these pigmented tumours are discussed, and it is concluded that they arise either from the pigment cells in the nerve that are derived from the retinal pigment (neurogenic melanoblasts) or from the chromoblasts (mesodermal melanoblasts) that grow into the optic nerve with the *lamina cribrosa*.

OTO-RHINO-LARYNGOLOGY.

Œstrogen and Otosclerosis.

E. P. FOWLER (*Archives of Otolaryngology*, December, 1948) states that it has been known for many years that certain sex-linked episodes frequently coincide with the onset or an increase in the deafness of otosclerosis and suggests that the sex hormones, in particular the œstrogens, play a part in these happenings. He points out that it is well known that some of the glands of internal secretion are mediating factors in bone metabolism. In experiments on animals the œstrogens have shown profound effects on the metabolism of bone. Several metabolic and endocrine disorders of bone may involve the otitic capsule, and in some respects resemble the lesions of otosclerosis, although all these disorders show distinctive morphological features which permit their differentiation. Neither do any of these diseases involve the otic capsule alone as does otosclerosis. The endochondral bone of the labyrinthine capsule remains from the second year of life in its original structural form, while many primitive embryonic features persist. Experimentally, the œstrogens increase calcification of bone and they effect the growth of bone more than do the androgens. The growth at the epiphysial-diaphysial junction in particular is curtailed or restrained. Administration of œstrogens to animals has been shown to cause replacement of the marrow cavities with endosteal bone throughout the skeleton, with the exception of the bone close to the *symphysis pubis*. The author asks why, if variable sensitivity to œstrogens can develop in the pelvic bone, a similar effect should not occur in the temporal bone. Unless there is an idiosyncrasy even large variations in the sex hormone tides appear to cause no pathological changes in the bones. There may be pathological consequences in susceptible persons, however. Growth, pregnancy and lactation are periods requiring additional calcium supply. Growth, pregnancy, lactation and the menopause are periods of changing activity of the sex glands often accompanied with diseases or disorders of bone coincident with known changes in œstrogen levels. The deafness of otosclerosis frequently increases during these periods. It is suggested that variation in the œstrogen levels may be one of the reasons for the higher incidence of otosclerosis in females. The thesis is advanced that œstrogen is so closely associated with calcium and bone metabolism that it is permissible to assign to it a part in the causation of otosclerosis.

Carcinoma of the Naso-Pharynx.

C. C. FOX (*Archives of Otolaryngology*, October, 1948) states that carcinoma of the naso-pharynx has a high mortality rate because the disease is often not recognized until it has reached an advanced stage. A series of twelve cases is discussed. The histological diagnosis was anaplastic squamous cell carcinoma in six cases, transitional cell carcinoma in four, and anaplastic cell carcinoma in two. A high percentage of the cells were young, undifferentiated and therefore highly radiosensitive. The ages of the patients ranged from thirty-two to seventy-four years; eight were females and four males. The earliest symptoms are important, especially cervical adenitis, earache and/or some degree of deafness, hæmoptysis and nasal obstruction, especially if two or more of such symptoms are simultaneously present. Less frequent first symptoms are pain in the naso-pharynx or along the distribution of the fifth nerve, headaches, disturbances of swallowing and post-nasal secretion. One unproductive search of the naso-pharynx should not be considered final, as it is surprisingly difficult to detect the early cancerous changes. Early cancer tissue is difficult to distinguish by inspection from hypertrophied lymphoid tissue. Later symptoms may be due to cranial invasion with involvement of one or more of the cranial nerves. In nine cases the cancer was first seen on the supero-lateral wall of the naso-pharynx, in two in the fossa of Rosenmüller, and in one on the posterior wall. The initial tumour later develops an ulcerated appearance with hæmorrhagic points and causes a foul odour. Metastasis usually occurs fairly early, first to the cervical lymph glands and next in frequency to the ribs, long bones, spine, skull, lungs, liver, kidneys and spleen. X-ray studies may reveal alteration, erosion or obstruction of the foramina, and invasion of the sphenoid bone and petrous part of the temporal bone.

Blast Perforation of the Ear Drum.

A. H. RICE (*Archives of Otolaryngology*, March, 1949) describes his experience with a series of 87 blast-damaged ear drums. The patients were examined soon after injury and were able to be followed for a considerable time afterwards. Blast was produced by violent explosion, such as V1 bombs and gun-breech explosions, often at very close range. An attempt was made to grade the severity of the drum injury by estimation of the area of drum missing and the area of ecchymosis; four grades were thus made, slight, moderate, severe and very severe. Hearing tests were able to be carried out only with the whispered voice and tuning forks. Generally speaking, when a patient was examined soon after injury there was a marked loss of hearing at the first examination. The hearing returned to normal over a period of two to four weeks. It was not determined whether the hearing defects were of conductive or of nerve type. As the speedy return of service personnel to duty was of paramount importance it was felt that the earliest application of a patch to fill in the defect was desirable. This series presented an opportunity to determine how early a patch could be used to advantage. Processed sheep's caecum was used throughout. The patch, cut to appropriate shape and size, was transferred from boracic solution to the perforation on a wire loop. When the edges of the perforation appeared pale and inactive, they were first treated with trichloracetic acid. In 42 cases the first patch remained in place; in 17 it was floated off by middle-ear drainage and was replaced one or more times. In 21 cases no patch was employed, in 17 of these because an aural discharge continued right up to the time of complete healing. In seven cases frank failure occurred. During the process of healing it was observed that Nature carried out its own *"débridement"*. It was found that a severely damaged drum required from five to twenty-one days to complete this process of liquefaction of blood clots, and absorption of ecchymoses and devitalized tissue. Any attempt at patching before this process was complete was doomed to failure, as the patch would be floated off by serous drainage. When the *"débridement"* was complete, the patch could be applied with good assurance of success. Some drainage of a thin, clear or whitish fluid, without evidence of inflammation in the ear, was noticed in most ears. Chemotherapy had no effect in reducing this discharge, which is thought to be non-infective. In seven cases unquestionable acute infection supervened, and in six of these the healing of the drum was most active, prompt and complete. Infection in these cases was controlled with penicillin or sulphadiazine. It is suggested that either the bacterial infection or the inflammatory reaction may stimulate healing activity. Of the whole series of 87 cases, failure to heal occurred in seven. Although failures were usually in more severely damaged drums, no definite prognosis can be given on this account, for some of the most severely damaged drums healed well.

Medical Matters in Parliament.

THE NATIONAL HEALTH SERVICE ACT.

THE SENATE.

IN the Senate on October 26, 1949, the HONOURABLE N. E. MCKENNA was given leave to bring in a bill for an act to amend *The National Health Service Act*, 1948. The Bill was presented and read a first time.

In moving that the Bill be read a second time, SENATOR N. E. MCKENNA said that the main purpose of the Bill was to amend Section 6 of *The National Health Service Act*, 1948. The text of the Act was published in this journal in the issue of February 5, 1948. Clause 6 is as follows :

> 6. The regulations may make provision for and in relation to the establishment, maintenance and conduct of a scheme for the payment by the Commonwealth, on behalf of persons who have received professional services from medical practitioners who are for the time being participants in the scheme, of the prescribed proportion of the fees prescribed in respect of those services.

Senator McKenna said that in its present form Section 6 provided that the regulations might make provision for the payment by the Commonwealth on behalf of persons to whom professional services had been rendered by medical practitioners participating in the scheme. The payment would be a prescribed portion of the fee prescribed for the particular purpose. The proposal was that the Commonwealth would pay 50% of the fee directly to the doctor. In the framing of the regulations it had become apparent that with Section 6 in its present form payment for medical services would be rigidly and literally limited to those items which were expressly referred to in schedules of the regulations. It was not considered feasible to draw up a complete and comprehensive schedule to meet all circumstances. Moreover, it was doubtful whether any set of regulations based on Section 6 in its present form could provide for the exclusion of certain patients such as compensation cases and patients living in areas where other classes of medical services were provided, or permit the making of special arrangements if thought desirable where agreements for the provision of medical services existed between doctors and particular groups, such as friendly societies and industrial and other medical schemes. There was also some doubt whether Section 6 permitted a mileage allowance to be prescribd which would have regard to the actual mileage travelled by doctors in rural areas to render professional services.

Section 6 in the Bill before the House was as follows:

> 6. (1) The regulations may make provision—
>
> (a) for or in relation to the establishment, maintenance, conduct and operation of a scheme for the provision of sickness benefits or medical services by way of payments by the Commonwealth in respect of professional services rendered by medical practitioners;
>
> (b) in relation to any arrangements made by the Minister under sub-section (4) of this section; and
>
> (c) in relation to matters incidental to a scheme established, or arrangements made, under this section.
>
> (2) Without prejudice to the generality of the last preceding sub-section, a scheme established under this section may—
>
> (a) define the circumstances in which persons shall, or shall not, be entitled to benefits or services under the scheme;
>
> (b) provide for participation in the scheme by medical practitioners in respect of particular classes of professional services; and
>
> (c) provide that the maximum fees to be charged or received by medical practitioners participating in the scheme in respect of professional services to which the scheme applies shall be the fees fixed by, or assessed under, the regulations as in force from time to time.
>
> (3) A maximum fee fixed by the regulations shall not be reduced unless the Minister has given not less than three months' notice of the proposed reduction by notice published in the *Gazette*.
>
> (4) Where an arrangement exists between a medical practitioner and a society, body or person under which professional services are rendered by that medical practitioner (whether or not a scheme established under this section applies in relation to those professional services), the Minister may make arrangements for the provision by the Commonwealth of sickness benefits or medical services, in respect of those professional services, in lieu of the benefits or services under the scheme.
>
> (5) Where arrangements made by the Minister under the last preceding sub-section are in operation in relation to any professional services, a scheme established under this section shall not apply in relation to those services.
>
> (6) Nothing in this section authorizes any form of civil conscription.

Senator McKenna said that the measure before the Senate was designed to provide the flexibility which would ensure that the regulations would be drawn up so that the benefits contemplated would reach the people to the fullest extent. He drew attention to the clause which stated that three months' notice had to be given before any fee could be reduced. Section 4 of the Bill provided that Section 16 of the Act should be amended by omission of the word "advisory".

Section 16 with the omission of this word will read as follows:

"The Minister may establish such committees as he thinks fit for the purposes of this Act."

Clause 5 of the Bill amended Section 22 of the principal Act. The first amendment was by the omission of the word "and" where last occurring in paragraph (*e*). Paragraph (*e*) would then read:

> for the making and recovery of charges in respect of medicines, materials and appliances supplied in connexion with a national health service or in respect of the replacement or repair of any appliance so supplied;

An additional paragraph would be inserted as follows:

> (*ea*) for investing any court of a State with federal jurisdiction with respect to any matter arising under the regulations; and

SENATOR N. O'SULLIVAN said that the Opposition did not oppose the measure. It was rather unfortunate that the people had had withheld from them for so long a service to which they were justly entitled. The blame lay with the Government, which had an insatiable desire to regiment, to regulate, and to put its finger into affairs which could be much better managed without its interference. The Government seemed to be determined to make a political football of a measure which should transcend party political considerations. Members of the medical profession realized that sickness could have the most devastating effect on the lives of members of families. The profession was prepared to cooperate with the Government to provide an adequate service for the poor and needy, but at the same time doctors had made it perfectly clear that they would not sacrifice their freedom or allow themselves to be dictated to, and that they would not willingly place themselves under the thumb of the Government. The Government had ample power to ensure that the members of all professions should comport themselves in a manner consistent with their responsibilities; but this did not mean that professional men should lose their professional freedom. The Minister had not yet made any official or unofficial statement in reply to the statement of the British Medical Association. When the principal measure was before the Senate some time previously, members of the Opposition had suggested that the measure should be withdrawn and that the Government should confer with members of the medical profession, the pharmacists and nurses' organizations and friendly societies, so that a practicable scheme of medical benefit would be formulated. If the scheme was to succeed, the cordial cooperation of members of the medical profession was absolutely necessary. Senator O'Sullivan suggested that the Government should for once forsake its stiff-necked attitude of socialistic prejudice, in order that some practicable scheme might be devised.

SENATOR J. A. COOKE said that on at least six occasions the Government had approached the British Medical Association in an effort to obtain the cooperation which Senator O'Sullivan suggested would be readily forthcoming. The Government had been accused by Senator O'Sullivan of attempts to regulate members of the medical profession. A similar criticism of regimentation might be made of the Government because it required certain classes of professional people and others to comply with certain formalities of the *Audit Act* and other governmental regulations. Professional people were often required by law to complete certain forms and to make certain declarations, but no one

would suggest seriously that the enforcement of such regulations was an interference with the freedom of the people. On previous occasions Senator Cooke had pointed out that when regulations were first introduced to require doctors to notify public health authorities of cases of contagious or infectious disease, they had aroused a great deal of opposition from members of the medical profession. However, medical science had advanced, and doctors did not hesitate to report cases of the prescribed types of disease. Senator Cooke believed that the majority of medical practitioners in the country believed in the soundness of the Government's scheme. However, those who controlled the British Medical Association had behind them the support of what was probably the strongest union in Australia. The activities of that union were completely uncontrolled. The present measure provided that the Government would pay 50% of medical charges of any person who required medical attention. This was a just provision, and whether the British Medical Association cooperated or not, Senator Cooke felt sure that the Government would obtain sufficient voluntary cooperation from medical practitioners throughout Australia to implement the scheme. The Government had done everything possible to obtain the cooperation of the British Medical Association, and it intended, within the provisions of the Constitution, to provide a free medical service for many persons who normally would not be able to afford such treatment.

SENATOR W. J. COOPER pointed out that the doctors had not evaded the law, but had quite legitimately applied to the High Court for an interpretation of the law. It was quite evident that the disagreement between the British Medical Association and the Government was due to the fact that the medical profession considered that the scheme would be the forerunner of a fully nationalized medical service. Senator Cooper referred to Section 22 of the principal Act, dealing with the power to make regulations. He said that the Director-General of Health, by power delegated from the Minister, could at any time issue regulations that would completely revolutionize the medical service. Senator Cooper referred to a booklet entitled "A National Health Service", which had been issued by the British Medical Association. This publication set out measures for a national health service aimed at the maintenance of health, the prevention of disease, the cure of disease and the upkeep of public hospitals, sanatoriums, mental hospitals and homes for the infirm. It suggested that a national health service could be conducted by the existing friendly society and other organizations, and proposed the adoption of a system similar to the Medical Benefits Schemes that had been instituted in New South Wales, Queensland and South Australia for people in the middle income groups. The suggestion was worthy of the close investigation of the Government.

Senator N. E. McKenna, in reply, said that Senator O'Sullivan was suffering from a very high degree of election fever. The catch-cry of "regimentation" would convince nobody, and would make no contribution to the content of the Bill. It was not true that the Bill had been made necessary by the recent decision of the High Court. The measure merely sought to amplify the existing Act. Senator McKenna also wished to join issue with the statement that the Director-General of Health would be able to effect drastic changes in the medical organization of Australia merely by making regulations under powers delegated by a Minister. The High Court decision gave clear authority for the assertion that not even the faintest element of compulsion could be used in relation to either doctors or dentists, even in procedural matters, much less matters involving the rendering of services. At any rate, the High Court had done some good by giving its decision. It had confounded those who had been proclaiming the absurd proposition that the Government aimed at the nationalization of the medical and dental professions. The Government knew that the friendly societies provided fairly comprehensive medical services for their members, and there were also many industrial medical benefit schemes, some of which operated on a contributory basis. The Government did not ignore the existence and the value of such schemes, and in the Bill it sought power to negotiate with the controlling body, with a view to reaching an agreement that would be satisfactory to them, to the doctors and to itself. When the Bill became law, regulations dealing with the Medical Benefits Scheme would be promulgated. Despite observations to the contrary by certain spokesmen of the British Medical Association, there would not be the slightest interference with the freedom of any patient to choose his own doctor; there would be no interference as to the form of treatment that the doctor should accord to his patient, and no interference with the relationship between doctor and patient. Senator McKenna then referred to a broadcast, which he said had been made throughout Australia by a member of the Federal Council of the British Medical Association. He directed particular attention to the statement that the Government asserted a right to know what the treatment was for—the nature of the patient's illness or disease. Senator McKenna said without reservation or hesitation that the broadcast either betrayed the most abysmal ignorance of an unpardonable type or was a deliberate misstatement. After further reference to this broadcast, Senator McKenna said that he had invited the British Medical Association to appoint a working committee to sit down with his officers and investigate their suggestions and those of the Government. They had declined to do so, unless first of all the Government was prepared to give way on every point at issue. In other words, they were prepared to confer under conditions which would leave nothing on which to confer.

The question was resolved in the affirmative and the Bill was read a second time.

In the committee stage Senator W. J. Cooper asked Senator McKenna why the word "advisory" had been omitted from Section 16.

Senator N. E. McKenna, in reply, said that the word "advisory" limited the functions of the committees. It appeared to the Government that it might want to establish various professional committees upon which some executive authority would be conferred. That would not be possible if the word "advisory" remained.

The Bill was agreed to.

The Bill was reported without amendment and the report was adopted.

The Bill was read a third time.

THE HOUSE OF REPRESENTATIVES.

The National Health Service Bill, 1949, was received from the Senate, and on the motion of the HONOURABLE E. J. HOLLOWAY, Minister for Labour and National Service, was read a first time.

THE HONOURABLE E. J. HOLLOWAY then moved:

That the Bill be now read a second time.

Mr. Holloway introduced the Bill to the House in a speech which was almost identical with that delivered by Senator the Honourable N. E. McKenna, Minister for Health and Social Services, in the Senate.

THE RIGHT HONOURABLE SIR EARLE PAGE said that there was no country whose national health had ever been dealt with in such a shameless, cavalier and callous fashion as had the national health of Australia by the Government. After a brush with Mr. Deputy Speaker, Sir Earle Page said that there were only two sections in the principal Act which really mattered—Section 6 and Section 22. If these two sections were removed, there would be little left of the principal Act. The Bill before the House would completely alter the framework of the National Health Service Scheme. He referred to a statement by the Minister for Health in regard to the keeping of clinical records of patients by medical practitioners for checking purposes. He also referred to the statement of the Prime Minister, Mr. Chifley, that all a doctor was asked to do was to furnish a return to show what the patients had been attended for. He was called to order by the Deputy Speaker and informed that he had to confine himself to the Bill before the House. Eventually Sir Earle Page was ordered to resume his seat.

The question was put. The House divided, and the question was resolved in the affirmative.

The Bill was read a second time.

The Bill was considered in committee.

Clauses 1 and 2 were agreed to.

THE HONOURABLE E. J. HARRISON discussed Clause 3 (scheme of sickness benefits or medical services). He said that the clause was designed, not so much to give effect to provisions contained in it, as to provide the Government with election propaganda. It was designed to encourage doctors voluntarily to come into the scheme, so that the Government might be able to offer the people a semi-free medical scheme on the eve of election. The legislation and Clause 3 particularly were evidence that there was still disagreement between the Government and the members of the medical profession. It appeared that the Government intended to rush the Bill through Parliament and immediately to gazette regulations so that it would be able to say to the people on the election eve: "This is part of the record on which we stand." The regulations that would be made under the Bill to fix the fees charged by medical practitioners for their professional services would strike at the very heart of the medical profession. He asked whether a newly qualified doctor was to be allowed to charge for his services the same fee as that of a surgeon of many years of experience. Was the fee of a specialist to be the same as that of a lad who had

just left the medical school? If that was so, what incentive would there be for doctors to specialize in certain branches of surgery or medicine or to do post-graduate work? The whole thing was ridiculous. Despite the "blah" in which the Government would indulge during the election campaign, the scheme would be received with the same coldness as the "free bottle of medicine" scheme had been received.

THE RIGHT HONOURABLE SIR EARLE PAGE said that the view of almost every medical practitioner was that the matters which, under the proposed new Section 6, would be dealt with by means of regulations, should be stated specifically in the legislation. If the Government really desired that a national health service scheme should operate in Australia, sooner or later it would have to reach agreement with the medical profession. The doctors would not be parties to a scheme that was controlled by regulation. A regulation to which medical practitioners might object might remain in force for many months before Parliament met and honourable members had an opportunity to submit a motion for its disallowance. Again, medical practitioners contended that the relation between doctor and patient was personal, intimate and confidential, and they refused to submit their clinical records to other persons for scrutiny. The British Medical Association had been accused of having adopted an uncooperative attitude, but that kind of allegation was completely false and wicked. The Minister for Repatriation (Mr. Barnard) would admit that the British Medical Association had assisted the Parliamentary Joint Committee on Social Security in every possible way, and Sir Earle Page drew the attention of the House to the fact that Mr. Barnard nodded his head in assent. Sir Earle Page urged the Government to turn for advice to the medical practitioners, who had at heart the improvement of the health of the people of Australia.

THE HONOURABLE T. W. WHITE said that with a great deal of its common cunning the Government had put into the legislation the short provision that: "Nothing in this section authorizes any form of civil conscription." At the same time the Bill prescribed the most severe penalties for the most trifling offences. How could the Government claim that the Bill did not still impose civil conscription on doctors? The Bill was just part of the socialist plan. In Sub-section (2) (c) of the proposed new section, reference was made to maximum fees to be charged or received by medical practitioners. It appeared that the Government intended to establish prices control of surgical operations and of medicine. Mr. White was not a constitutional lawyer, but he thought that the Government had not the constitutional power to do so. The Government had lost the referendum on prices control, and yet it intended to lay down the maximum price that a doctor might charge for an operation.

MR. A. V. THOMPSON said that no new principles were involved in the Bill. Its purpose was solely to make it possible for those doctors who were prepared to cooperate with the Government to implement the health scheme. Members of the Opposition were opposing the Bill merely for opposition's sake.

THE HONOURABLE E. J. HARRISON said that it was clear that the Bill was a price-fixing measure. If maximum fees were to be fixed for various forms of treatment, medical practitioners would not be encouraged to do the post-graduate work that a specialist had to do. The Government would not be able to induce self-respecting medical men who had the interests of their patients at heart to participate voluntarily in the scheme.

THE HONOURABLE E. J. HOLLOWAY, Minister for Labour and National Service, said that the proposed new Section 6 would not alter materially the principles already agreed to. One reason for its introduction was that it would give the Minister for Health and the officials of the Department of Health greater opportunities to consult with members of the medical profession and with organizations such as friendly societies. Another reason was that fear had been expressed that when the medical profession and Commonwealth departmental officers had reached agreement regarding the fees that might be charged for certain kinds of medical treatment, the scale of fees might be changed by the Commonwealth without notice having been given to the medical profession of the change. The new section was designed to ensure that the agreed scale of fees should not be altered without three months' notice of alteration being given. The clause had no other purposes. Mr. Holloway referred to the contention that the records of patients would be subject to inspection by departmental medical practitioners for the purpose of checking accounts. He said that the only way in which such records would be available would be in the event of a dispute between a patient and a medical practitioner. And such a dispute might not occur in a million years.

The committee divided on the question of the clause. The clause was agreed to.

After the temporary chairman had declared the question resolved in the affirmative and Clauses 3, 4 and 5 agreed to, Mr. Bernard Corser protested that the division did not resolve the question, because fewer than twenty-five members voted on either side. The temporary chairman upheld the objection, and the committee resumed its consideration of Clauses 3, 4 and 5.

THE HONOURABLE T. W. WHITE referred to the handbook "A National Health Service" issued by the British Medical Association, and said that the Association had offered a practical scheme that would be much better than the one proposed by the Government.

THE HONOURABLE H. C. BARNARD remarked that some of the health proposals submitted by the British Medical Association were identical with the recommendations of the Parliamentary Joint Committee on Social Security. He hoped that Parliament would pass the Bill, and that the British Medical Association and the medical profession generally would see the advantage of accepting the Government's proposal.

THE RIGHT HONOURABLE SIR EARLE PAGE asked the Government not to make regulations under this measure, but to let the people decide the matter on December 10 at the general election. He was satisfied that when they returned to the chamber in the next Parliament, his party would have a majority and the measure would be repealed. No regulations associated with it would ever be made, because they would bring into being a system that would not only improve the health of the people, but would also use every voluntary agency, such as friendly societies and doctors' and nurses' organizations, and that would steadily improve public health and give cause for greater pride than ever in what had been achieved.

The clause was agreed to.

Clauses 4 and 5 were agreed to.

The title was agreed to.

The Bill was reported without amendment, and the report was adopted.

On the motion of the HONOURABLE E. J. HOLLOWAY the Bill was read a third time.

Correspondence.

A CASE OF HIDRADENOMA OF THE VULVAL REGION.

SIR: Regarding the article and comments on the case of hidradenoma of the vulval region by Mr. Bruce Boscence, I would like to say:

1. That the correct pre-operative diagnosis in such cases depends upon experience and would have been enhanced had the references been read before operation rather than after the pathologist's report.

2. The operation as performed was inadequate had the pre-operative clinical diagnosis—epithelioma of the vulva—been correct.

Yours, etc.,
LESLIE GLEADELL, F.R.C.S., F.R.C.O.G.

14 Parliament Place,
Melbourne, C.2,
December 9, 1949.

OVERCROWDING THE MEDICAL PROFESSION IN NEW SOUTH WALES.

SIR: It is surprising that the article by Hugh R. G. Poate on overcrowding of the medical profession in New South Wales (THE MEDICAL JOURNAL OF AUSTRALIA, December 18, 1948) has created so little interest, both in the ranks of the profession generally and, as far as can be judged, among the representatives of the various State Branches of the British Medical Association. Dr. Poate's findings are of great importance. The most striking is that the University of Sydney Medical School is one of the few such bodies in the English-speaking world which does not limit enrolments—the University of Queensland seems to be the only other one.

Dr. Poate states that in 1948 in New South Wales there were 11·0 doctors per 10,000 of population, and that in 1953 there will be 16·78—a 50% increase in ten years. A similar

situation exists in Queensland. Prior to 1940 all Queensland's medical practitioners came from schools elsewhere. In the ten years 1940-1949, 300 medical graduates left the new Queensland Medical School, and were rapidly absorbed into practice. Information available from university authorities shows that the number of graduates to be expected in 1950 is about 55, in 1951 about 80, and in 1952 about 60. It is to be noted that the last group will have spent the final year of their course in residence, so that in 1953 about 140 new doctors will have to find employment, compared with a normal figure of 30. Judging by the number of undergraduates in the lower years, these alarmingly large figures will be maintained for some time in the future.

A few years ago, Mr. Colin Clark declared that 52 graduates were needed each year to provide for Queensland's growing medical needs. Present trends of practice, plans for the provision of medical benefits and the stream of post-war migrants may have invalidated this figure. On the other hand, the Commonwealth Reconstruction Training and Financial Assistance Schemes, State scholarships and fellowships, and the recently announced Commonwealth aid scheme (whereby 400 scholarships a year have been allocated to Queensland) are likely, unless action is now taken, to keep the number of undergraduates in medicine at an undesirably high level.

That an unnecessarily large number of medical students is not desirable must be quite apparent. With the present swollen years, facilities are proving inadequate, the quality of instruction is impaired, failures are at a high level, and the opportunities for teachers to participate in research are reduced to vanishing point. Again, public money is spent on training students who will never graduate, on producing more graduates than are needed to provide the community's medical services, and on maintaining unduly large teaching units.

The increasing interest shown by State and Federal Governments in education is to be applauded. We look forward to the day when education to the highest level will be available to all, and nobody will be debarred because of inadequate financial means.

While everybody should be entitled to receive tertiary education, however, the economic demands of society must regulate the number to be admitted. Let the Government provide secondary education for all students whose parents cannot support them, but let it assist to the tertiary level only those students, selected on a competitive basis, who are best qualified to fulfil the wants of the various professions.

The time is ripe for the new Federal Government, in consultation with State and municipal health departments and universities, to make a survey of Australia's need for medical practitioners to safeguard national health, and to ensure that the supply may meet the demand in such a way that the best interests of the community will be served.

Yours, etc.,

Brisbane, "SENEX."
January 2, 1950.

SOME PROBLEMS ASSOCIATED WITH THE MANAGEMENT OF CARCINOMA OF THE BREAST.

SIR: Dr. Ham accuses me of deciding against radiotherapy on the basis of one case. He thus makes an accusation in which there is no truth. Probably his offence was not deliberate but caused by his reading only the first and last paragraphs in my letter. A parent springing to the defence of an only child may be partly forgiven for overlooking fairness. As I pointed out, I decided against radiotherapy, partly through the exercise of my own reason and partly on the basis of many thousands of cases, recorded and analysed by earnest investigators, backed by the resources of great clinics, which have no prejudice either for surgery or radiotherapy. And his term "obsolete"—when I bring the data up to 1949! "Inaccurate and misleading"—when I try my best to quote accurately and reason logically!

Let me request patient instruction from Dr. Ham.

"Has there been any fundamental improvement in radiotherapy over the last fifteen years?" We are concerned with prophylactic radiotherapy. Dr. Ham quotes two "revolutionary" improvements, first "in the estimation of tumour doses" and second in "the knowledge of factors influencing the biological effect".

Firstly, "estimation of tumour doses". There is no tumour. How can the "revolutionary" estimation be applied to the hypothetical cell of unknown whereabouts? That Dr. Ham should have chosen this as an example of fundamental improvement illustrates the difficulty of his search. Let us consider ground chosen by Dr. Ham, the cancer cell of intermediate sensitivity in the subcostal lymphatics or internal mammary glands on the side of operation. What would be the dose delivered to such a cell at an unknown point between clavicle and hypochondrium? What would be the dose given to a similar cell of exactly known whereabouts, in narrow field irradiation of a visible metastasis in the sternoclavicular region? Can you deliver a certainly cancericidal dose to this cell of unknown whereabouts in the subpleural lymphatics and avoid damage to the lung and to the thin post-operative skin? Dr. Ham has already tried to answer similar questions. But he has not yet made the matter clear. He used the conditional and subjunctive moods, with their "mights" and "wouldst", gave no physical units, not even arithmetical ratios, and fell back upon his success in removing clinical evidence of tumour, although this success had not been questioned. May I presume upon his patience and ask him to supplement his previous answers by dealing with the above questions.

Secondly, "revolutionary changes in . . . the knowledge of factors influencing the biological effect of radiation". What are these mysterious factors? How does a knowledge of them affect the treatment? How have they made the X rays so much more cancericidal in the last fifteen years that the change may be termed "revolutionary"? At least I know that Dr. Ham is not referring to the importance of vascular supply and of varying sensitivity, because these factors have been recognized for much more than fifteen years.

Dr. Ham says that it is a pity I do not see the many recurrences in the radiotherapy clinics. But I can quite imagine them. Even in the surgical clinics they are sometimes seen. I saw some last week. There were none in the operative field, the part which Dr. Ham would have treated prophylactically, and this is my usual experience. They were in the axilla of the opposite side, in the thorax of the opposite side (my associate aspirated a litre of fluid from this patient) and in the lumbar vertebræ, all parts which Dr. Ham would not have irradiated in any case. Let us suppose, however, that the surgeon has sown the wound with cancer cells, either through his faulty case-selection or his faulty operative technique. Can a dose of X rays be delivered to every point in the axilla and on the chest wall under the thin skin flaps, so that cancer cells of intermediate resistance will certainly be killed at whatever point they may happen to lie? What is the dose per unit volume of tissue given under these circumstances? What is the exact ratio of this dose to the dose applied in the case with tumour, which Dr. Ham so often falls back upon?

This question may be easier. What is the ratio of the dose given to the supraclavicular glands in prophylactic post-operative radiotherapy, and in radiotherapy given when the glands are involved by metastases evident clinically?

Dr. Ham hints that there is something significant in my quotation of American work. What does Dr. Ham mean? Does he mean that American radiotherapy is bad? Such a general statement would be easy to make and hard to substantiate. I could just as easily (and with evidence provided by Dr. Ham) make similar hints about the surgery in that city where McWhirter's radiotherapy flourishes. Since the days of Coolidge, and before, the Americans have made outstanding contributions to the design and manufacture of X-ray machines, and today they are not backward in the pure and applied science of physics. Does Dr. Ham seriously suggest that they cannot use the machines which they build so well? The great journals published by the American radiologists and radiotherapists are the world's largest. Are their contents of poor quality? It would be strange that America should be backward in only one branch of medicine, and that radiotherapy. Are you serious, Dr. Ham?

Dr. Ham prefers British authorities (perhaps because McWhirter is British, and McWhirter favours radiotherapy). So, let us to London. Noel Newton (now a registrar at University College Hospital) wrote to me from this city on June 16, 1949, that "McWhirter's attack with simple mastectomy and radio-therapy to glands is frowned on very much in London". I think Dr. Ham has earned the reproof of Socrates: "To you, it seems, it makes a difference who the speaker is, and from what country he comes; you do not merely consider whether the fact be or be not, as he states it."

Dr. Ham mentions that Haagensen's operation takes five hours. They have found at the Presbyterian Hospital and Columbia University, New York, that the short operation (one hour) gives twice the recurrence rate and half the cure rate. Perhaps in Edinburgh they use the short operation? Halsted took six hours to do the radical operation on the breast, and it is his operation. Cushing, his pupil, operated with similar painstaking thoroughness. So did

Kocher. It might also be remarked that if a patient needs resuscitation after a radical mastectomy there has been something wrong with the operation. Blood has been wasted, or the operation has been too rough, or both.

Another point about which Dr. Ham and I are concerned is our anxiety lest the general practitioners should be misled. One method by which they may be misled and confused is by the determined mixing up of tumour areas with non-tumour areas, and tumour cases with non-tumour cases.

But it is with each of my questions that I am most concerned. I would fain be taught.

Yours, etc.,
V. J. KINSELLA.

235 Macquarie Street,
Sydney,
December 10, 1949.

TRAUMA AND TUMOURS.

SIR: As it is generally accepted that innocent uterine tumours (for example, fibroids) are, we think, of the commonest human body tumours, and that uterine malignancy, which is, similarly, an extremely common cancer, have, as their part-presumable cause, "trauma" (childbirth or the injury of chronic inflammation), it therefore occurred to us that simple and malignant brain tumours in childhood (a very common growth) could also be partially caused by injury or trauma. Our statistical researches tend to show this, but as we are conducting a world-wide survey on the incidence of such diseases in children (brain cancer), our results may take some time to complete. The use of forceps during delivery, prolonged labours, precipitate birth *et cetera* may be a causative factor. We are seeking this information, too.

Yours, etc.,
LEIGHTON O. D. WEBER, M.B., B.S. (Sydney).
BETTY YVONNE WEBER.

3 Blyth Street,
Brunswick, N.10,
Victoria.
December 20, 1949.

The Royal Australasian College of Physicians.

EXAMINATION FOR MEMBERSHIP.

AN examination for membership of The Royal Australasian College of Physicians will take place in May and June, 1950. The written examination will be held in capital cities of the Commonwealth where candidates are offering. The clinical examination will be held in Sydney. Only those candidates whose answers in the written examination have attained a standard satisfactory to the Board of Censors will be allowed to proceed to the clinical examination.

The dates of the examination will be as follows: written examination (capital cities), Saturday, May 20, 1950; clinical examination (Sydney), during the week commencing June 19, 1950.

Applications to appear before the Board of Censors should be made in the prescribed form and must be in the hands of the honorary secretary of the College not later than Saturday, April 22, 1950. Candidates should signify in which city they desire to take the written examination. Application forms are obtainable from the honorary secretary, 145 Macquarie Street, Sydney.

SIMS COMMONWEALTH TRAVELLING PROFESSOR.

IT is announced by the Presidents of the Royal College of Physicians of London and The Royal Australasian College of Physicians that Professor D. M. Dunlop, of Edinburgh, has been appointed the Sims Commonwealth Travelling Professor for 1951. Professor Dunlop, who occupies at present the chair of therapeutics and clinical medicine at the University of Edinburgh, has had a most distinguished career and includes amongst his degrees and diplomas those of F.R.S. (Edinburgh), B.A. (Oxon.), M.D. (Edinburgh) and F.R.C.P. (Edinburgh).

It is expected that Professor Dunlop will arrive in Australia about April of 1951 and will visit both the Dominion of New Zealand and the various States of the Commonwealth of Australia.

The Sims Commonwealth Travelling Professorship was endowed in 1947 by Mr. Arthur Sims and family, of Christchurch, New Zealand, with the primary object of furthering the advancement of medical science by the interchange of qualified medical persons between the component parts of the United Kingdom and the British Commonwealth.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 99, of December 22, 1949.

AUSTRALIAN MILITARY FORCES.

Royal Australian Army Medical Corps.

SX700004 Captain (Temporary Major) R. N. McCann relinquishes the temporary rank of major and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (4th Military District), 14th October, 1949.

To be Temporary Major, 5th August, 1949.—VX700077 Captain T. H. Hurley.

ROYAL AUSTRALIAN AIR FORCE.

Citizen Air Force: Medical Branch.

The appointment of Temporary Squadron Leader C. H. Walsh (267801) is terminated on demobilization, 8th November, 1949.

Reserve: Medical Branch.

The appointment of Flight Lieutenant T. B. Ready (257669) is terminated, 24th February, 1946.

Australian Medical Board Proceedings.

TASMANIA.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Act*, 1918, of Tasmania, as duly qualified medical practitioners:

Beazley, Gerald Patrick, L.L.M., R.C.P. and S., 1941 (Ireland), New Norfolk.
Woolley, Vincent Guy, M.B., B.S., 1949 (Univ. Melbourne), Launceston.
Taranto, Robert, M.B., B.S., 1949 (Univ. Melbourne), Launceston General Hospital.
Horton, John Affleck, M.B., B.S., 1949 (Univ. Melbourne), Launceston.
Elrick, William Lindsay, M.B., B.S., 1949 (Univ. Melbourne), Royal Hobart Hospital.
Roche, Mark Francis, M.B., B.S., 1949 (Univ. Melbourne), Royal Hobart Hospital.

NEW SOUTH WALES.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1938-1939, of New South Wales as duly qualified medical practitioners:

Au, Kee Hock, L.M.S., 1928 (Univ. Singapore), c.o. Royal Prince Alfred Hospital, Camperdown.
London, Michael Solon, M.B., B.Ch., 1941, D.A., 1949 (Univ. Witwatersrand, South Africa), 81 Darling Point Road, Darling Point.
Murphy, Thomas Roy, L.R.C.P., L.R.C.S. (Edinburgh), L.R.F.P.S. (Glasgow), 1935, Yambo Street, Morisset.
Rimington, Ronald Arthur, M.B., B.S., 1946 (Univ. Melbourne), 24 Hampden Road, Artarmon.
Walker, Oliver Nyma, M.B., B.S., 1911 (Univ. Durham), c.o. Commonwealth Health Department, Sydney.

The following additional qualifications have been registered:

Boxall, John Stephen, 20 Kiora Road, Miranda (M.B., B.S., 1936, Univ. Sydney), M.R.A.C.P., 1948.

Paterson, Robert Francis, 143 Macquarie Street, Sydney (M.B., Ch.M., 1926, Univ. Sydney), D.L.O. (R.C.P. and S.), 1929 (England).

Rawle, Ronald Maxwell, 143 Macquarie Street, Sydney (M.B., B.S., 1930, Univ. Sydney, F.R.C.S., England), F.R.A.C.S., 1948.

Sharp, Henry Walter Ritchie, 139 Macquarie Street, Sydney (M.B., B.S., 1935, Univ. Sydney), D.D.M., 1948 (Univ. Sydney).

Shortridge, Dennis Thorman, Carrington Street, Wahroonga (M.B., B.S., 1938, Univ. Adelaide), Dip. Ophth. (Univ. Sydney), Dip. Ophth. (Univ. Oxford), D.O.M.S., 1947, F.R.A.C.S., 1948.

QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Acts, 1939 to 1948*, of Queensland, as duly qualified medical practitioners:

Abrahams, Ellis William, M.B., B.S., 1941 (Univ. Melbourne), M.D., 1946 (Univ. Melbourne), M.R.C.P., 1947 (London), c.o. Department of Health and Home Affairs, William Street, Brisbane.

Evans, Cyril Percival Victorious, M.B., B.S., 1943 (Univ. Sydney), c.o. Dr. G. Whyte, Esk.

Groves, Garry John, M.B., B.S., 1949 (Univ. Melbourne), Mater Hospital, South Brisbane.

Thatcher, Ralph Arnold, M.R.C.S. (England), L.R.C.P. (London), 1935, M.R.C.O.G., 1948 (England), c.o. Dr. H. S. Waters, City Mutual Building, Queen Street, Brisbane.

Woodley, John, M.R.C.S. (England), L.R.C.P. (London), 1940, D.A., R.C.P. and S. (England), 1947, 27 Mayfield Street, Ascot, Brisbane.

Allen, Lucius, M.B., B.S., 1949 (Univ. Queensland), 47 Abbott Street, Ascot, N.E.2, Brisbane.

Barnett, Robert Reid, M.B., B.S., 1949 (Univ. Queensland), 14 Barlow Street, Brisbane.

Bowly, Frank Sutcliffe, M.B., B.S., 1949 (Univ. Queensland), Benaraby, Lilly Street, Sherwood, Brisbane.

Brightman, Maxwell Esmond Juel, M.B., B.S., 1949 (Univ. Queensland), Warrawee Street, Toowong, S.W.1, Brisbane.

Brown, Edward Harry, M.B., B.S., 1949 (Univ. Queensland), Brook Street, Kalinga, Brisbane.

Buckley, John Herbert, M.B., B.S., 1949 (Univ. Queensland), 106 Wilston Road, Newmarket, Brisbane.

Byth, John Gordon, M.B., B.S., 1949 (Univ. Queensland), 19 Lindsay Street, Ashgrove, Brisbane.

Cantamessa, Remo, M.B., B.S., 1949 (Univ. Queensland), Box 116, Proserpine.

Chapman, Douglas John, M.B., B.S., 1949 (Univ. Queensland), Stanley Terrace, Taringa, Brisbane.

Cheesman, Edgar Neville, M.B., B.S., 1949 (Univ. Queensland), Abuklea Street, Wilston, Brisbane.

Chester, Brian Charles Michael, M.B., B.S., 1949 (Univ. Queensland), Sandon Street, Graceville, Brisbane.

Cilento, Ruth Yolanda, M.B., B.S., 1949 (Univ. Queensland), Seriano, 683 Ipswich Road, Annerley, Brisbane.

Clarke, Alexander Howard Burnett, M.B., B.S., 1949 (Univ. Queensland), 505 Brunswick Street, New Farm, Brisbane.

Clarke, Charles George Drury, M.B., B.S., 1949 (Univ. Queensland), 22 Windermere Road, Ascot, Brisbane.

Clouston, Alexander David, M.B., B.S., 1949 (Univ. Queensland), 47 Bramston Terrace, Herston, Brisbane.

Duhig, John Taylor, M.B., B.S., 1949 (Univ. Queensland), Hunstanton, L'Estrange Terrace, Kelvin Grove, Brisbane.

Dyer, Henry Paul, M.B., B.S., 1949 (Univ. Queensland), 6 Inkerman Street, Woolloongabba, Brisbane.

Francis, John Brenton, M.B., B.S., 1949 (Univ. Queensland), 34 Creswick Street, Clayfield, Brisbane.

Friend, William Douglas, M.B., B.S., 1949 (Univ. Queensland), Yaroon Street, Gladstone.

Galea, Eugene George, M.B., B.S., 1949 (Univ. Queensland), Liaveena, Elliott Heads, Bundaberg.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED DECEMBER 17 1949[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.[2]	Australian Capital Territory.	Australia.[3]
Ankylostomiasis	*	1(1)	1	..	1	..	..	..	3
Anthrax	*	..	..	..	..	..	..	..	..
Beriberi	*	*	*	*	*	*	..	*	..
Bilharziasis	*	..	..	..	..	..	..	..	..
Cerebro-spinal Meningitis	3(1)	1(1)	..	..	..	..	..	..	4
Cholera	..	..	..	..	..	..	..	..	..
Coastal Fever(*a*)	*	*	..	*	*	*	*	*	..
Dengue	*	*	*	*	*	*	..	*	..
Diarrhœa (Infantile)	..	..	7(5)	..	..	..	..	..	7
Diphtheria	10(3)	4(4)	7(3)	..	4	..	..	..	25
Dysentery (Amœbic)	*	1(1)	4(4)	..	2	..	..	..	7
Dysentery (Bacillary)	*	..	..	..	..	..	..	..	..
Encephalitis Lethargica	..	1	..	..	..	..	..	..	1
Erysipelas	*	*	*	..	*	*	..	..	..
Filariasis	*	..	..	..	..	..	..	..	..
Helminthiasis	*	..	*	*	*	*	*	*	..
Hydatid	*	..	*	*	..	*	*	..	..
Influenza	*	*	*	..	*	*	..	*	..
Lead Poisoning	*	*	..	*	*	*	*	*	..
Leprosy	..	..	..	..	..	..	..	..	..
Malaria(*b*)	*	..	1	..	..	..	..	..	1
Measles	*	*	*	69	*	*	..	..	69
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	10(1)	17(7)	..	42	..	2(1)	..	..	71
Psittacosis	*	..	*	..	*	*	*	*	..
Puerperal Fever	..	..	..	..	..	..	..	..	..
Rubella(*c*)	*	*	2	*	..	*	..	..	..
Scarlet Fever	46(24)	18(6)	8(3)	5	5	2(1)	..	..	84
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	*	..	1	*	1	*	*	*	2
Trachoma	*	..	*	*	*	*	*	*	..
Tuberculosis(*d*)	49(43)	12(10)	4(2)	3	7	9(4)	..	1	85
Typhoid Fever(*e*)	..	..	..	4	..	..	..	..	4
Typhus (Endemic)(*f*)	..	..	..	..	..	..	..	..	..
Undulant Fever	..	..	..	..	..	..	..	..	..
Weil's Disease(*g*)	*	*	..	*	*	*	*	*	..
Whooping Cough	*	*	*	38	*	*	..	..	38
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] The form of this table is taken from the *Official Year Book of the Commonwealth of Australia*, Number 37, 1946–1947. Figures in parentheses are those for the metropolitan area.
[2] Figures not available.
[3] Figures incomplete owing to absence of returns from the Northern Territory.
* Not notifiable.
(*a*) Includes Mossman and Sarina fevers. (*b*) Mainly relapses among servicemen infected overseas. (*c*) Notifiable disease in Queensland in females aged over fourteen years. (*d*) Includes all forms. (*e*) Includes enteric fever, paratyphoid fevers and other Salmonella infections. (*f*) Includes scrub, murine and tick typhus. (*g*) Includes leptospiroses, Weil's and para-Weil's disease.

Geaney, Bruce, M.B., B.S., 1949 (Univ. Queensland), Baroona, Howard Street, Rosalie, Brisbane.

Gillies, William Elliott, M.B., B.S., 1949 (Univ. Queensland), 62 Heidelberg Street, East Brisbane.

Honours.

NEW YEAR HONOURS.

IN last week's issue an unfortunate error was made in the announcement of the knighthood conferred by His Majesty the King on Dr. Thomas Ernest Victor Hurley. Dr. Hurley has been created a Knight Commander of the Most Excellent Order of the British Empire.

Public Health.

POLICE OFFENCES (AMENDMENT) ACT, 1908, AS AMENDED: RESTORATION OF AUTHORITY.

THE following notice has appeared in the *New South Wales Government Gazette*, Number 232, of December 23, 1949.

It is hereby notified, for general information, that the Authority of Dr. David Herbert Lewis, of Lawson, to procure and be in possession of drugs for the purpose of his profession, which Authority was withdrawn on and from the 24th May, 1940, has been restored on and from the 19th December, 1949.

C. H. MATTHEWS.

Notice.

CRICKET MATCH AT SYDNEY.

THE annual cricket match between teams representing the British Medical Association and the Australian Dental Association in New South Wales will be played at the Sydney Cricket Ground (Number 2) on Wednesday, February 8, 1950. The match will commence at 10.30 o'clock a.m. Medical men wishing to play are asked to communicate with Dr. F. M. Farrar, Sydney Hospital.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Hales, Ian Barnewall, provisional registration, 1949 (Univ. Sydney), 2 Jersey Road, Artarmon.

Watson, Keith Gordon, M.B., B.S., 1948 (Univ. Sydney), 21 Parriwi Road, Mosman.

Hay, Judith Alison Ruth, provisional registration, 1949 (Univ. Sydney), Parramatta District Hospital, Parramatta.

Fogl, Henry Walter, provisional registration, 1949 (Univ. Sydney), 206D Victoria Road, Bellevue Hill.

Hunter, Irvine John, provisional registration, 1949 (Univ. Sydney), 7 Woodside Avenue, Lindfield.

Woodhouse, Edward Beaumont, M.R.C.S. (England), L.R.C.P. (London), 1935, 43 Gerrish Street, Gladesville.

Winkworth, Alan Charles Stepney, M.B., B.S., 1948 (Univ. Sydney), 19 Kylie Avenue, Killara.

Medical Appointments.

Dr. T. E. Lowe has been appointed a member of the Anti-Cancer Council of Victoria, in pursuance of the provisions of Section (1) (b) of the *Anti-Cancer Council Act*, 1936, of Victoria.

Dr. N. C. Joel has been appointed quarantine officer, Bunbury, Western Australia, in pursuance of the provisions 1937 *to* 1948, of Queensland.

Dr. M. J. J. O'Reilly has been appointed Deputy Director, Laboratory of Microbiology and Pathology, Department of Health and Home Affairs, in pursuance of the provisions of *The Public Service Acts*, 1922 *to* 1948, of Queensland.

Diary for the Month.

JAN. 14.—Queensland Branch, B.M.A.: Council Meeting.
JAN. 16.—Federal Council of the B.M.A. in Australia: Meeting in Melbourne.
JAN. 17.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JAN. 25.—Victorian Branch, B.M.A.: Council Meeting.
JAN. 28.—Queensland Branch, B.M.A.: Council Meeting.
FEB. 1.—Western Australian Branch, B.M.A.: Council Meeting.
FEB. 1.—Victorian Branch, B.M.A.: Branch Meeting.
FEB. 2.—South Australian Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135 Macquarie Street, Sydney): Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester United Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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